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ON

DISEASES OF THE CHEST:

BEING

CONTRIBUTIONS TO THEIR CLINICAL HISTORY, PATHOLOGY, AND TREATMENT.

PART I.

DISEASES OF THE LUNGS.

PART II.

DISEASES OF THE HEART, AND THORACIC ANEURISM.

BY

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PREFACE.

It has been my object, in the following pages, to depict disease as I have found it at the bedside, and to illustrate the application of therapeutics to thoracic affections by details of cases. The work is in nowise a systematic treatise on Diseases of the Chest, but a series of contributions — chiefly in the form of lectures — to the clinical history, pathology, and treatment of some of its more important affections. My views on some points in the pathology of the lungs will be found to differ from those of many authors. On this, I will merely remark that the opinions which I have expressed have not been arrived at without careful and prolonged examination of both the healthy and morbid lung-tissue.

Hope Street, Liverpool,
March, 1868.

82295

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PLATE I.

FIG. 1.

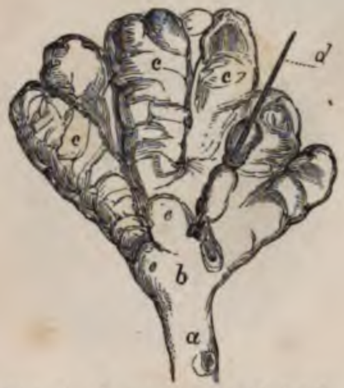


Fig. 1 is a drawing of a terminal bronchial tube, with a group of air-sacs, or lobulette, connected with it, divided transversely (human). *a*, the terminal bronchial tube; *b*, the dilated extremity of the terminal bronchial tube; *c c c*, individual air-sacs. At *d*, a bristle is seen passed into an air-sac; one end is seen opening into the common cavity in which the bronchial tube terminates. At *e e* are seen the openings of other sacs which lie beneath those which are exposed; six sacs are seen converging to the common centre. The markings in the air-sacs denote the boundaries of the alveoli.

FIG. 2.

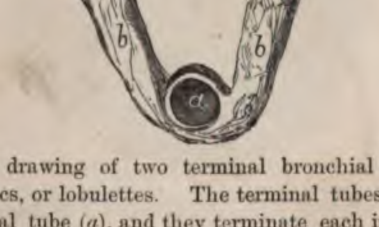


Fig. 2 is a drawing of two terminal bronchial tubes, with two groups of air-sacs, or lobulettes. The terminal tubes are seen to pass from a bronchial tube (*a*), and they terminate each in a group of air-sacs. *b*, terminal bronchial tube; *c c c*, air-sacs; *d d*, openings leading to air-sacs beneath those exposed. This diagram was taken from the lung of an infant under one year of age.

PLATE II.

Fig. 3.

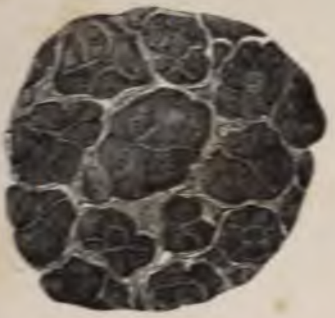


Fig. 3 represents a very thin slice of a cat's lung (injected, inflated, and dried), from the surface of the lung. The eye is looking upon the cut surface. The depressions, *a a*, are the bottoms of the air-sacs, resting on the pleura. The light coloured lines that surround them are their walls, and the small depressions seen within the walls, *b b*, marked off by less distinct lines, are the alveoli. The specimen from which this drawing was made was a very good one, and the drawing may be considered as fairly representing the appearance presented.

Fig. 4.

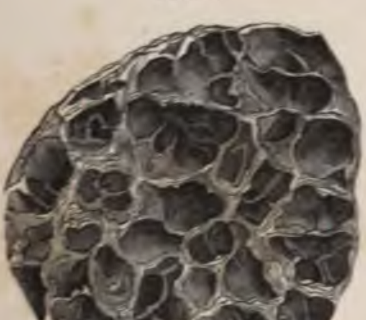


Fig. 4 is a drawing of a similar slice to that of Fig. 3, taken from a human lung. *a a a* are the air-sacs, *b b* the alveoli. The lighter portions represent the walls of the air-sacs and the alveoli.

PLATE III.

Fig. 5.

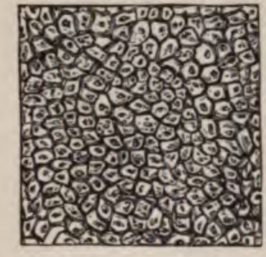


Fig. 6.

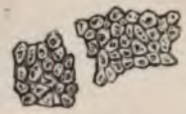


Fig. 5 shews the appearance of the epithelial cells lining the air-sacs, highly magnified, and treated with acetic acid. Some of the centre cells were drawn with the aid of the camera lucida, their outline being traced as thrown down on the paper.

Fig. 6. Epithelial cells lining the air-passages of the pigeon, highly magnified. Drawn with the aid of the camera lucida

Fig 7.

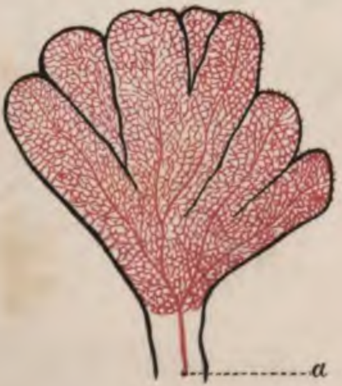


Fig. 7 shews the arrangement of the pulmonary plexus in the air-sacs of the lung. *a*, branch of pulmonary artery.

PLATE IV.

Fig. 8.

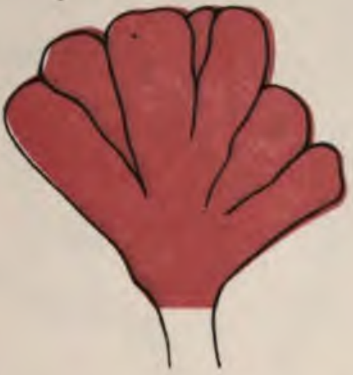


Fig. 8 represents the air-sacs distended with exudation. (Pneumonia — stage of hepatization.)

Fig. 10.

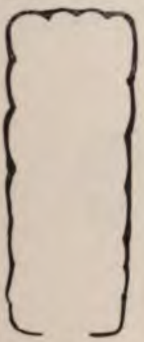


Fig. 9.

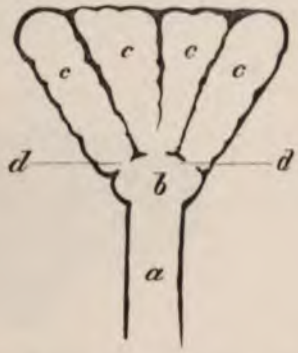
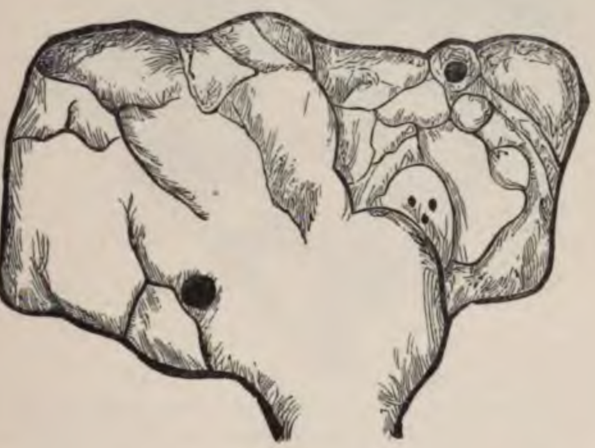


Fig. 9 represents a terminal bronchial tube with a group of air-sacs; *a*, terminal bronchial tube; *b*, dilated extremity of the terminal bronchial tube forming the *point de réunion* of the air-sacs; *c c*, air-sacs; *d d*, mouths of air-sacs.

Fig. 10 represents a single air-sac.

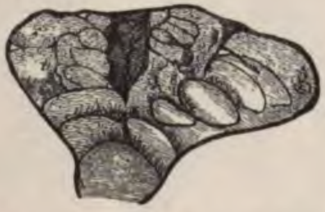
PLATE V.

FIG. 11.



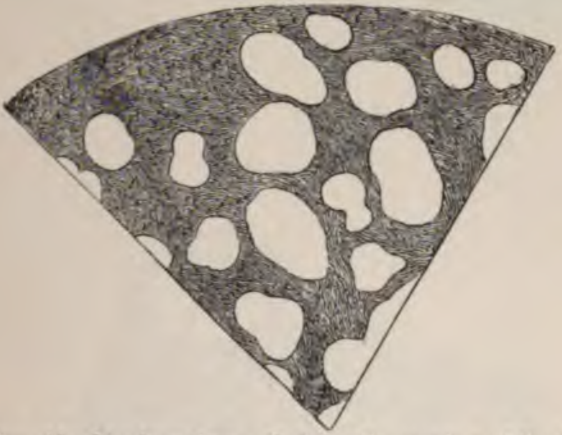
Air-sacs of a piece of emphysematous lung, injected, inflated, and dried The lung from which the piece was taken was emphysematous along its margins. The piece was taken from the margin. It shews dilatation of the air-sacs, more or less obliteration of the alveoli, and slight perforation. The black spots represent the perforations. The air-sacs in the piece of lung examined varied in diameter from 1-20th to 1-30th of an inch — being more than double their ordinary size in health.

FIG. 12.



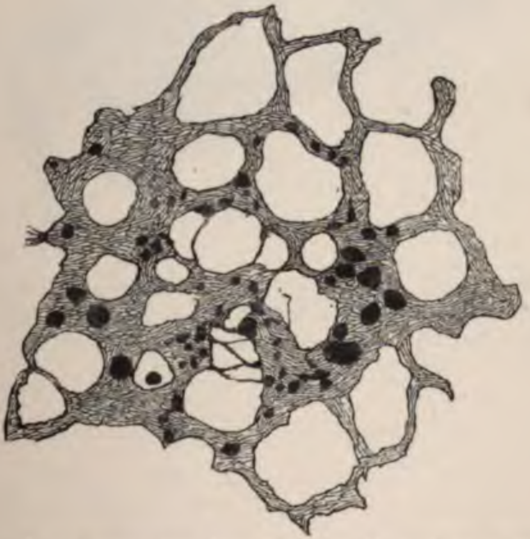
Air-sacs from the same lung as Fig. 11, but not emphysematous. The alveoli are shewn with their septa well marked. The diameter of the sacs in the part examined varied from 1-45th to 1-70th of an inch. Figs. 11 and 12 were both taken by means of the camera lucida, and the same magnifying power was used in both. They therefore shew the relative size of healthy and emphysematous air-sacs.

FIG. 13.



A slice of healthy lung-tissue. It shows the openings of the divided air-sacs, and the walls separating them, in a healthy condition. No perforations are seen. (Taken by means of the camera lucida.)

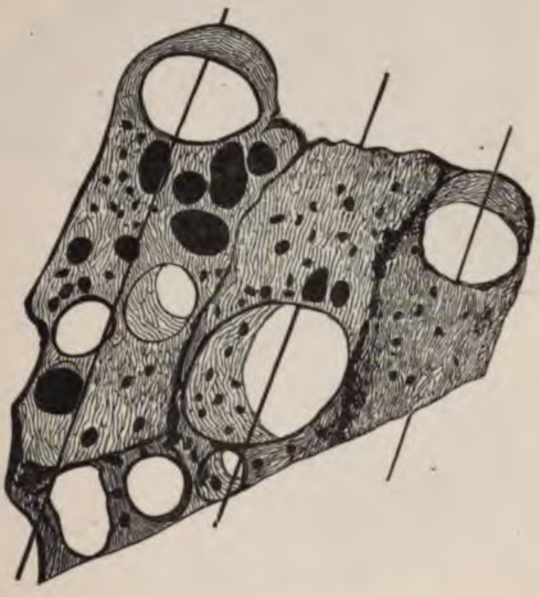
FIG. 14.



Slice of emphysematous lung-tissue in the earlier stage of the disease. The black spots show the perforations in the walls of the air-sacs. (Taken by means of the camera lucida.)

PLATE VII.

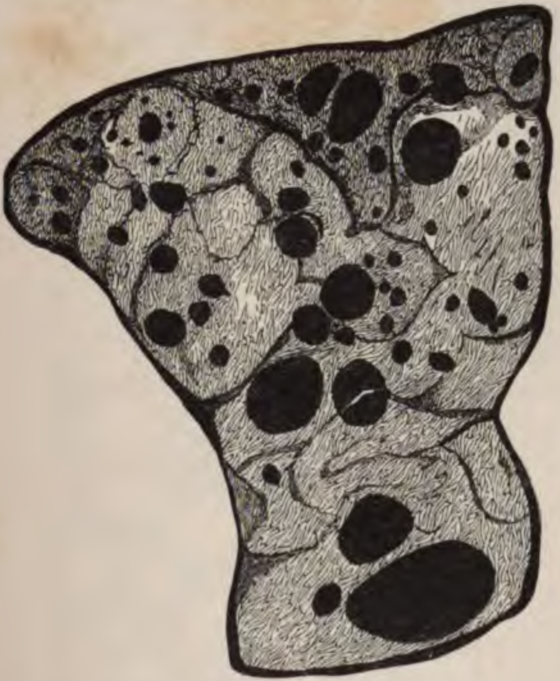
Fig. 15.



Air-sacs of emphysematous lung, as seen through dissecting microscope. The bristles are passed through the sacs. The upper walls of two sacs, and the lower wall of one sac, are seen with their perforations.

PLATE VIII.

Fig. 16.



Taken from a portion of lung very emphysematous. It shows the appearance of the air-sacs in a dilated condition, with their partitions much ruptured, and full of large perforations. (Drawn by means of the camera lucida.) The black spots shew the perforations. The cavities — the distended and broken-down air-sacs — in the piece of lung from which this was taken had a diameter of 1-16th, 1-20th, and 1-27th of an inch.

PLATE IX.

Fig. 17.

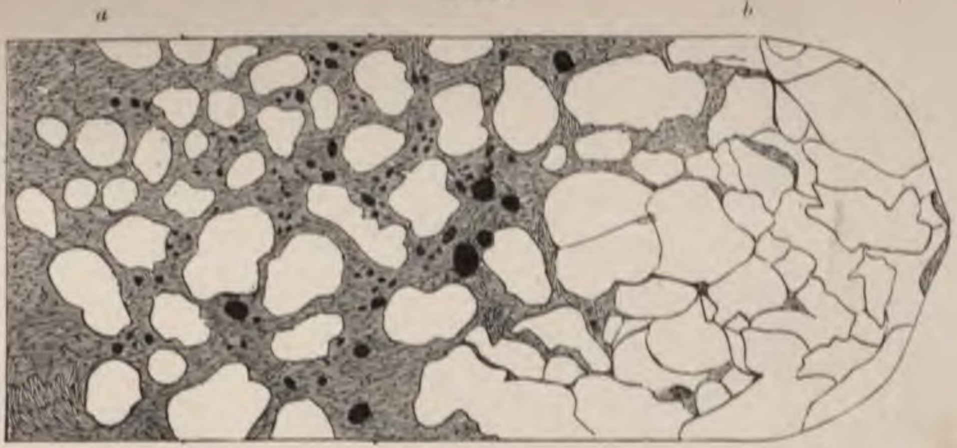


Fig. 17 shews the appearance of a piece of highly emphysematous lung, taken from the margin of the lung, where a distinct bulging or "vesicle" existed. The white spaces denote the openings of the divided air-sacs; the black spots shew the perforations of their walls; and the lighter parts represent the walls themselves. At *a*, the lung-tissue is tolerably healthy; at *b*, the walls of the air-sacs are broken up, and the membranous shreds and septa alone remain. (Taken by means of the camera lucida, and magnified to one-half the linear dimensions of Figs. 11, 12, 13, 14, and 16.

PART THE FIRST.

DISEASES OF THE LUNGS.

CHAPTER I.

OBSERVATIONS ON THE ANATOMY OF THE LUNGS.

The bronchial tubes of the lungs, after several divisions and sub-divisions, which are, for the most part, dichotomous, terminate in a dilatation, into which opens a number of cavities, which constitute the ultimate expressions of the air-tubes. These cavities, to which various names have been given, I have elsewhere* called *air-sacs*, as being, in my opinion, more appropriate to their shape and arrangement than any term previously used; and the series of air-sacs connected with the extremity of each bronchial twig, &c., I have called a *lobulette*.

Each *lobe* of a lung is composed of a number of *lobules*, the outlines of which can be seen on the surface of the lung. A *lobule* is made up of a series of *lobulettes*, and thus the description of a single *lobulette* will suffice for that of the entire *lobule*.

Each *lobulette* consists of a collection of air-sacs, varying in number from six to twelve. The air-sacs are somewhat elongated cavities, communicating with the dilated extremity of a bronchial tube by a circular opening, which is usually smaller than the sac itself, and has sometimes the appearance of a circular hole in a diaphragm, or as if it had been punched out of a membrane stretched across the entrance to the sac. When this is the case, the sac dilates suddenly beyond the opening. The sacs of the lobulette are placed side by side, and are separated from each other by thin membranous walls. Their shape, when properly inflated, or when distended by some material which has set in them, as gelatine, or a mixture of wax and turpentine, is polygonal. They approach the circular in form; but, in consequence of mutual pressure, the parietes become somewhat flattened. The sacs increase slightly in size as they pass from the bronchial tube to their fundus, the latter being usually the broadest part of the sacs; but they often have an almost uniform diameter throughout. All the sacs pass from the extremity of the bronchial tube *towards* the circumference of the lobule of which the lobulette forms a part; they consequently radiate from the tip of the bronchial twig. The sacs connected with one lobulette do not communicate with those of another lobulette. As the sacs pass towards the boundary of the lobulette, they often bifurcate; and here and there circular orifices exist, leading to smaller air-sacs. If we trace the air-sacs from their fundus, we may say that, passing from the periphery of the lobulette, and diminishing somewhat in size, they all terminate in the dilated extremity of the bronchial tube; as they thus proceed, they often join, two or three together, and these terminate in a single mouth. The tube which results from the union of two or more sacs is smaller in capacity than the sacs taken together, but greater than either of them individually. The dilated extremity of the bronchial tube, above alluded to, constitutes the *point de réunion* of all the air-sacs, and may be considered as the common centre of the lobulette. The air-sacs of the adult human lung vary in diameter from 1-45th to 1-85th of an inch. (For illustrations of this paragraph, see Plate I., figs. 1 and 2.)

---

* "The Anatomy of the Human Lung," page 135.

The walls of which the air-sacs are composed are exceedingly thin, and much sacculated; *i. e.*, they have in them a number of small, shallow, cup-like depressions, separated from each other by portions of membrane, which are more or less raised, and project into the anterior of the sacs. The bottom of an air-sac presents the same appearance as its lateral walls; and the cup-like depressions, or *alveoli*, are there very numerous. The number of these alveoli varies very much; I have counted as many as ten at the fundus of an air-sac in a cat's lung; in the human being I have counted five and six, but the number is generally somewhat less. I have found the entire number in each air-sac varying from eight to twenty. (See Plate II., figs. 3 and 4.)

The air-sacs rest, externally, by their fundus, on the pleura; but within the substance of the lung they in part rest on, and are supported by, the bronchial tubes and blood-vessels.

The air-sacs are separated from each other by thin walls, the membrane composing which, in a lung inflated and dried, is very transparent. The projections of this membrane in the shape of thin processes, having a sharp margin, constitute the septa between the alveoli; and wherever an opening exists leading into a smaller sac, this membrane projects in a similar way, and forms a circular orifice which is much smaller than the cavity to which it leads:—the sac, in fact, dilates abruptly on the distal side of the opening. It is in the membrane composing these walls, and in the septa of the alveoli, that the capillaries of the pulmonary artery are spread out.

As I have before remarked, the number of air-sacs belonging to a lobulette varies: I have counted as many as six communicating with a bronchial tube incised horizontally, so that probably only half the sacs were left; this, however, is a larger number than is usually found; from six to eight or ten is the more common number.

Each lobulette is separated from those by which it is surrounded by walls, which appear to resemble in every way the walls of the air-sacs; and in an adult inflated and dried lung, careful observation is necessary to make out the partitions. That perfect septa do exist, is proved by laying open, in a recent lung, a bronchial tube, to its ultimate division; when, by placing a fine blowpipe in it, and blowing down it, a single lobulette is alone inflated.

The separation of the lobulettes is further distinctly perceptible in the recent lungs of infants, in which the line of demarcation between the lobulettes is often plainly seen, on the surface.

The observation of the fœtal lung, however, affords most satisfactory evidence of the separation of the lobulettes, and tends to confirm the views here taken of the arrangement of the ultimate air-tubes.

The recent lung of a full-grown fœtus presents on its surface no appearance of air-sacs, vesicles, or cells; but if it be inflated, it will present different appearances, according as the inflation has been partial or entire. In a portion of lung only partially inflated, a number of tubes will be seen terminating beneath the pleura in cæcal extremities, their light colour contrasting strongly with the surrounding dark-coloured tissue. The exact arrangement of these tubes may be sometimes observed. They will be found to exist in groups or clusters, and to pass from the cæcal ends to a point, in which they terminate, and where they all appear to join; or, to describe them in the inverse direction, they pass from a point at some distance from the surface, and radiate towards the pleura. The tubes are seen to have numerous constrictions and bulgings; they terminate in extremities rounded, or nearly so. In a preparation of this kind it is often easy to see the bronchial tube for a short distance before it terminates; and not only is the terminal group of air-sacs (lobulette) visible, but two or more of the previous ones arising laterally from the bronchial tube may be also seen. The uninflated lung-substance lying between the distended air-sacs is distinctly seen when there has been only slight inflation; and the isolated condition of each group of sacs is very apparent; the sacs passing from different points radiate in different directions. In a lung which has been fully inflated, the grouped appearance is lost, and the ordinary condition of the distended lung is observed.

If, in a fœtal lung, we follow out a bronchial tube, we find that the smaller branches of the tube have, connected with them, clusters of little pyriform red-coloured bodies, which look very much like a number of grapes attached to their stalks. In a fœtus of six months, I have found it somewhat difficult to separate each individual body, but in a full-grown fœtus there is no difficulty in doing so; each little body is attached to a short pedicle. If air be blown down a bronchial tube leading to the exposed bodies, the latter become distended.

The little bodies just described are the ready-formed groups of air-sacs, or lobulettes; the pedicle with which each is connected is the terminal bronchial twig. The lobulette thus formed is surrounded by its sheath, and no communication exists between it and adjoining ones.

That the appearances I have described in the artificially inflated fœtal lung are not the result of any abnormal distension, I have been able to prove by observing the same appearances in the lung of a child in which only partial respiration had taken place.

Different opinions have been expressed as to whether the air-sacs communicate with each other by any orifices except that by which they communicate with the bronchial tube. Adriani states that he has observed such orifices, and specially mentions that they are most clearly to be seen in the stag. Dr. Thomas Williams considers that the "intercellular passages" intercommunicate, and are perforated by secondary passages at every point. Rossignol, Schultz, Mandl, and Milne-Edwards deny the existence of such communications. From observations, made with much care and frequently repeated, I have satisfied myself that the opinion expressed by the latter authors is correct. I have never found, either in the lungs of man or in those of the dog, cat, pig, sheep, or any other mammal I have examined, any lateral orifices of communication between the sacs of a lobulette.

As I have before mentioned, a number of the lobulettes constitutes a lobule. These lobules are of various sizes; they are each surrounded by a membranous wall, which is dense and strong, allowing of no communication between the blood-vessels passing to adjoining lobules. The lobules are connected together by a small quantity of areolar tissue, in which the branches of the pulmonary veins take their course.

The termination of each bronchial tube has a particular and special character. It differs in its anatomical arrangement from the other portions of the tube, and, no doubt, differs also in function; it constitutes, in fact, a part of the true respiratory system. As this condition of the bronchial tube has important physiological and pathological bearings, I shall refer to it in detail.

Rossignol was the first who pointed out the existence of a structure, at the termination of the bronchial tubes, similar to that which exists in the air-sacs. He says: "In the bronchial divisions of the two last, and sometimes three last orders, it is plainly seen, when they are opened longitudinally, that their surface is covered over, or, as it were, honeycombed, by a number of small regular shallow cavities, placed side by side, and separated by thin perfect walls of the same height, which project into the interior of the bronchial tube."

The existence of these bronchial *alveoli* has been noticed by subsequent observers. They may be easily seen in a lung injected, inflated, and dried, and sometimes even in one which has been simply soaked in spirit for a few days. They resemble the *alveoli* of the air-sacs; they consist of little cup-like cavities, resting upon the bronchial tube, and opening into its cavity; they are surrounded by walls, and their shape is more or less oval, or circular, quadrangular, or polygonal. In a preparation in which the pulmonary artery has been injected, branches are seen to pass from that vessel to these alveoli, before it reaches the *air-sacs*.

These *alveoli* are best seen in the lungs of some of the lower animals. In the cat they are especially distinct, and very easily recognised; in this animal they are found in the last ramifications of the air-tubes and their dilated extremity. In man, however, I have never seen them except at the extremity of the tube, and in many lungs I have examined, I have found no appearance of them at all. This is to be explained by the fact that these *alveoli* diminish in number with advancing age. It would seem that as the lungs become more distended, these depressions or cavities become more and more obliterated. In the infant I have found the alveoli in the last bronchial tubes and their dilated extremities, but I have not found them in the penultimate and earlier branches, and even in the last branches they do not always exist previous to the dilatation. When the *alveoli* are found in the tube previous to the dilatation, they do not generally commence abruptly, but, being thinly scattered at first, their number gradually increases. As far as my observations go they are very rarely to be found in the adult, and never in the aged, except in the terminal dilatation.

It will be seen that my observations, as to the tubes in which these *alveoli* are found, do not quite accord with those of Rossignol, who places them in the two or three last divisions of the tubes, whereas I have never been able to find them except in the last. It seems to me probable that Rossignol has drawn his conclusions from observations on the lungs of the lower animals, in some of which, as, for instance, the cat, they exist to a greater extent than in man.

The walls of the air-sacs and alveoli are formed of yellow elastic fibrous tissue, a basement membrane, and an epithelium, together with the capillary vessels constituting the pulmonary plexus. The fibres of the elastic tissue are arranged in bundles, and singly. They are found surrounding the mouths of the air-sacs, running through their walls in various directions, and encircling the alveoli. The fibres traversing the walls are placed at some distance from each other, so that spaces are left between them where the blood-vessels are uncovered, except by the basement membrane and epithelium. At the circumference of the alveoli, and at the margins of the sacs, the fibres are often gathered into bundles of considerable size.

It is to this tissue that the elasticity of the air-sacs, and of the whole lung, is due; and the changes which take place in it, in one of the diseases to be hereafter considered, constitute a most important feature of the affection.

The basement membrane is thin, homogeneous, and transparent. It is a prolongation of that lining the bronchial tubes; but it becomes finer and more delicate as it passes from the latter vessels into the air-sacs.

Some difference of opinion exists as to the presence of an epithelium on the walls of the air-sacs. From careful and repeated examination of a very considerable number of specimens of the lungs of man, and of various mammals, I have satisfied myself of the existence of such an epithelium. No doubt whatever can be entertained, that the ciliated columnar epithelium, so characteristic of the bronchial mucous membrane, ceases at the commencement of the alveoli — the true respiratory portion of the lungs; for in no instance, in which proper precautions have been taken to avoid errors, have I seen an epithelium of such a character present in the air-sacs. It seems to me altogether unnecessary to attempt to prove, from analogy, that an epithelium *ought* to be absent or present in the true pulmonary system, for if the fact be capable of demonstration, no theoretical argument against it can ever avail, and none in proof of it is required.

If a piece of the fresh lung of any mammal be taken, the pleura carefully stripped from it, and a small portion of the pulmonary tissue be removed and washed, and then placed under the microscope, an appearance will be observed not very unlike that which is presented by the free surface of a serous membrane. The portion of tissue examined will be found covered with a number of small flattened bodies, of various sizes and shapes, many of them approaching the circular, but most of them being polygonal; beneath these bodies, in different parts, will be seen bands of yellow elastic fibres and capillary blood-vessels. The flattened bodies are tolerably distinct, but the outline of many of them is faint; nuclei are apparent in a large number of them. (See Plate III., figs. 5 and 6.)

Round the sides of the piece of tissue under examination a number of detached bodies, and also small granules, will be seen, resembling those just described. If now acetic acid be added, the outline of these bodies becomes more distinct, and the nuclei more apparent. These bodies have all the characteristics and appearances of epithelial cells, and such they seem to me to be. They have not quite the same appearance as those of a serous membrane; they are smaller, and more rounded in shape, and less distinct in outline, but in the possession of nuclei (which are visible in all after the addition of acetic acid) and granular matter, they resemble the ordinary pavement epithelia.

The cells form a distinct and complete coating to the walls of the air-sacs and alveoli, and they rest immediately upon the basement membrane. My measurements have given from $\frac{1}{6000}$ to $\frac{1}{3000}$ of an inch as the diameter of these cells. In the cat, I have found them varying from $\frac{1}{5000}$ to $\frac{1}{2000}$ of an inch; in the sheep, from $\frac{1}{5000}$ to $\frac{1}{3750}$ of an inch.

Although, as I have previously stated, demonstration is better than analogy, yet, with reference to the existence of any particular structure of the human body, it becomes of the utmost importance to ascertain how the parts which have a similar function to perform in other animals are constituted; and especially does this apply where investigation can only be carried on by means of the microscope. I have frequently observed a pavement epithelium present in the lungs of reptiles; and the existence of an epithelium in the air-passages of the bird seems to me to be so obvious, as not to leave the slightest doubt of the fact. Under a magnifying power of 360 I have seen the cells perfectly distinctly. They are very small, and present at first a very faint outline, but, on the addition of acetic acid, their outline comes clearly into view, and their nucleus is rendered very apparent. On the gills of fishes an epithelium of a similar character is also easily recognised.

In examining these epithelia, a perfectly fresh lung should be used, for they seem soon to become destroyed, or at least to lose their characteristic appearance.

The capillaries of the pulmonary arteries, which form the pulmonary plexus, ramify in the membrane described above as forming the walls of the air-sacs and the septa of the alveoli. As the respiratory changes take place in the air-sacs, it is obviously of the greatest importance that the integrity of these latter should remain unimpaired, for any loss of structure they may undergo diminishes the amount of aerating surface over which the blood has to pass. The pathological bearings of these points will be dwelt on hereafter.

THE BLOOD-VESSELS OF THE LUNGS. — *The Pulmonary Arteries.* — The branches of each pulmonary artery, on entering the lungs, attach themselves to the bronchial tubes and their divisions, and accompany them throughout their course as far as their termination. This arrangement presents no exception in any part; the number of branches of artery is equal to the number of bronchial tubes; but at the termination of those tubes, where the air-sacs commence, a difference of arrangement exists.

Although every bronchial tube has its accompanying artery, yet these are not always given off at the same level as the tubes themselves. The vessels pass from the main trunk at obtuse angles; many of them have to pass partly round the air-tube which is the satellite of their trunk, to reach the tube for which they are destined.

Each branch of a pulmonary artery, as it accompanies a bronchial tube, distributes its branches in like manner with the tube itself, and thus often divides dichotomously; every lobule has its artery entering it, which at its entrance is a little less than the bronchial tube, but as the vessel gives off branches it gradually diminishes in size more rapidly than does the tube, so that at the termination of the latter the artery is not more than one-fifth the size of the tube.

From the second and third divisions of the arteries some small vessels are given off, which do not correspond with any divisions of the air-tubes. These small vessels pass off nearly at right angles, and may be traced passing to portions of the pulmonary substance, which lie in close contiguity with the arteries.

The branches of the artery are connected with the bronchial tubes, and with the lung substance, by means of areolar tissue, which is loose and somewhat coarse in the larger vessels, near the root of the lung; but in proportion as the vessels become smaller, so does the connection become more and more intimate, and the tissue of union closer in its character, so that, when the artery reaches its lobule, it is closely applied to the parts with which it is in contact.

When the artery reaches the termination of the bronchial tube it gives off a number of branches, which are distributed to the air-sacs; before doing so, however, branches are derived from it which pass to the alveoli situated at the termination of the tube; these latter branches terminate in plexuses which are formed in the alveoli, and are arranged in a similar manner to those which exist in the air-sacs. The branches destined for the air-sacs take their course in the walls which separate the sacs from each other; they do not pass in any very regular or definite manner, but run in different parts of the sacs, and in different directions, along their entire length. Many of them may be observed passing straight from the commencement of the sacs to their fundus, and this course is more or less observed by all. In whatever way the *arterioles* pass, they gradually diminish in size as they proceed to the fundus of the sacs, and this diminution results from the giving off of a number of lateral branches from both sides of the vessel; these branches pass off at right or obtuse angles, and run in the walls of the sacs, they pass round the circumference of the sacs, and unite with branches coming from other arterioles, so that an imperfect and irregular network is formed by these primary branches of the arterioles. Independently of these branches, the saccular arteriole furnishes a number of smaller ones, which almost immediately after their origin terminate in a capillary plexus. (See Plate III., fig. 7.)

The pulmonary plexus is formed by the branching of the vessels above described. It is placed in the walls which separate the air-sacs, in the septa between the alveoli, and around the margins of the openings which exist in the sacs. The membrane in which the plexus lies is exceedingly thin, and the plexus consists of a single layer of vessels, which in no instance is doubled on itself. If a single alveolus be examined, it will be found that the vessels are spread out throughout its entire extent, and that the plexus rises in the septa surrounding it. In these septa, however, as well as at the margins of the circular orifices alluded to, the vascular network does not reach quite to the free border of the membrane composing them; in injected preparations, a small portion of transparent uninjected tissue is seen beyond the extreme vessels.

There is, as a rule, no distinct and separate vessel for each alveolus, no definite plexus consisting of afferent artery and efferent vein, belonging to each "vesicle," as was formerly supposed. The sacs of each *lobulette* have their vessels, which run irregularly through their walls.

The plexus, when formed, maintains a tolerably uniform size throughout. In a well-injected preparation, inflated and dried, it will be seen that the spaces between the vessels are somewhat greater in diameter than the vessels themselves.

The branches of the pulmonary artery do not anastomose until they reach the termination of the bronchial tubes. In the air-sacs their branches anastomose freely. It is difficult to decide whether the vessels of one lobulette anastomose with those of another. In looking at a preparation, injected and dried, it seems as though the septum, separating one lobulette from another, resembled in every respect the other walls of the air-sacs; but, as the lobulettes are originally separate and independent bodies, as seen in the fœtal lung, it appears probable that the vessels of each are distinct. If so, the external walls of two adjoining lobulettes must have the capillary vessels of two sacs ramifying in them, or, in other words, there must be two walls brought in contact, with two layers of capillaries lying side by side.

This view militates against the general opinion, that in *no part* of the lungs is there more than a single layer of capillaries in connection with the aërating membrane, and that the air circulates on both sides of the plexus of vessels; but yet, from the mode of formation of the lobulettes, and from the examination of some preparations of the lung substance of the adult, in which I have traced areolar tissue passing between the lobulettes, and have been able to separate them partially from each other, I am induced to believe that each lobulette has its separate vessels, which do not communicate with those of the adjoining ones, but terminate in their proper radicle vein, and that thus the capillaries, which are placed in the outer wall of the lobulette, are only exposed on one side to the atmosphere.

Whatever be the case with regard to the vessels of the different lobulettes, it is quite clear that the vessels of one lobule do not anastomose with those of another lobule; for when a portion only of a lung is injected, and the injection-pipe is placed in a small branch of the pulmonary artery, it will be found that the injection has filled a number of lobules supplied by the artery; but, lying by the side of these, others will be seen, into which no injection has found its way; did the vessels of adjoining lobules anastomose, this would not be the case.

From the arrangement I have described, it will be seen that the pulmonary plexus is, for the most part, exposed to the air on both sides, a condition which is of the utmost importance with reference to the proper aëration of the blood. As the vessels lie in the walls of the air-sacs, and in the septa between the alveoli, the air circulates on both sides of them, in consequence of a single layer alone existing. This statement applies as a general rule, but if the opinion I have expressed above with reference to the external sacs of the lobulette be correct, then an exception exists to the double exposure of each blood-globule; and, moreover, there is another exception, viz., in the vessels which are placed at the fundus of those sacs which are situated at the periphery of the lobules. It is clear that when the fundus rests on the sheath which invests the lobule, and which is separated from the sheath of the adjoining one by areolar tissue, or rests on the pleura, the vessels which are spread out on the alveoli of the fundus can only be exposed to the air on one side.

It appears, therefore, from these remarks, that some portion of the pulmonary plexus is not so arranged that its capillaries shall have air circulating on both sides of them, but that by far the greater portion of it is thus placed. It may further be remarked that, in all probability, before the blood reaches the fundus of the sacs I have alluded to, or even the periphery of the lobulettes, it has passed over other alveoli, and been aërated in them, and is, in fact, ready to be taken up by the pulmonary veins.

The branches of the pulmonary artery, which are distributed to the bronchial alveoli, anastomose freely at the mouths of the air-sacs with the capillary plexus formed in the walls of the sacs. The vessels distributed to the sacs themselves, and the plexus arising from them, pass from the apex to the fundus of each sac, and there terminate in the radicles of the pulmonary veins; in consequence of this arrangement the blood must pass through a series of alveoli before it is taken up by the last-mentioned vessels, and thus it is exposed during a somewhat prolonged transit to the influence of the air. In all probability, each drop of blood, in whatever part of the lung it may circulate, is arterialised to the same extent, from the quality of the aërating surfaces over which it passes.

The Pulmonary Veins.—These vessels, receiving the blood from the plexus of the air-sacs, pass from the periphery of the lobulettes, and running in the spaces between the lobules, make their way, independently, to the root of the lung.

The Bronchial Vessels.—It has long been held that the bronchial arteries are distributed to the air-tubes, the areolar tissue, and the vessels of the lungs, and that they pour their contents *partly* into the pulmonary veins, and *partly* into certain deep bronchial veins, which have been described by most anatomists as accompanying the arteries within the lungs. An opinion has also been entertained that a communication exists between the bronchial vessels and the branches of the pulmonary arteries. Without referring to the experiments and results of other observers, I proceed to state my own.

My observations have been made on the lungs of the cat, the dog, the rabbit, the pig, the calf, and the sheep, as well as on those of man. The following remarks have special reference to the results obtained in the human lung.

When the pulmonary artery is injected so that the fluid reaches the pulmonary plexus but does not pass to any extent into the pulmonary veins, the blood-vessels of the bronchial mucous membrane and of the other portions of the bronchial tubes *never* become injected. When, however, the injection is continued so as to fill the pulmonary veins, the vessels of the bronchial tubes become partially injected.

When the pulmonary veins are injected, whether the pulmonary plexus be well filled or not, the deep vessels of the bronchial tubes and the vessels of the bronchial mucous membrane are always injected. The bronchial tubes are often seen to be injected when the pulmonary plexus is only very partially so, the fluid seeming to find its way from the pulmonary veins into the vessels of the bronchial tubes more readily than into the capillaries of the air-sacs.

In a preparation that has been well injected through the pulmonary veins, the whole vascular system of the lungs becomes filled, viz., the branches of the pulmonary veins, the capillaries of the air-sacs, the pulmonary arteries, together with the vessels of the bronchial tubes, blood-vessels, lymphatics, and areolar tissue.

When the bronchial artery, or a branch of it as it enters a lobe of the lung, is injected, the vessels of the bronchial tubes become injected, both those of the mucous membrane and of the deeper structures, and the fluid finds its way into the pulmonary veins, which may become filled. If the injection be continued, it is possible to inject the pulmonary plexus through the medium of the bronchial arteries, and I have often found in such cases injection in the branches of the pulmonary artery. In addition to the vessels of the bronchial tubes becoming injected, those of the coats of the blood-vessels and lymphatics, and of the areolar tissue, become so.

In injecting the bronchial artery I have always found a difficulty, in the human lung, in properly filling the vessels along the whole length of the mucous membrane. I have traced vessels nearly to the extreme end of the bronchial tubes; but I have found that the mucous membrane has been best injected nearest the spot where the injection-pipe was inserted; in fact, that although the larger vessels were filled very nearly to the extremity of the tubes, the finer ones were only partially so. This seems to me to be due to the fact that, throughout the entire extent of the tubes, there is so free a communication between the bronchial vessels and the pulmonary veins, that the fluid finds its way into the latter more readily than into the fine plexus of the extreme bronchial tubes, the vessels of which are very small.

When the vena azygos is injected, and the injection finds its way into the bronchial veins, the bronchial tubes do not become injected; and on examining the course of the vessels which have been filled with the injection, I have never, in the observations I have made, found the so-called deep bronchial veins, as *venæ comites* of the bronchial artery. I have always found a small vein, or veins, generally a single vessel, which accompanied the artery in its course along the bronchus; but it, or they, always appeared to me to terminate in the structures of the bronchus and lower part of the trachea, and in the bronchial glands at the root of the lung. I have examined a large number of lungs, both of man and the lower mammalia, and I have always failed to discover any veins within the lungs accompanying the branches of the bronchial arteries. I have injected with fine injection, such as I have used for injecting the pulmonary capillaries, the vena azygos, on different occasions; I have found the bronchial vein, or veins, at the root of the lung filled, and on examining the bronchial tubes, the bronchus, and trachea, I have found the first uninjected, except just at their commencement, the second well injected, and the lower part of the last more or less so. In injecting a bronchial artery which was fairly within the lung, I have never found any return of the injection through a bronchial vein. These facts lead me to conclude that the so-called deep bronchial veins do not accompany the bronchial arteries within the lungs, and do not return any portion of the blood which is supplied to the bronchial tubes, but that they are simply vessels which return the blood from the structures situated about the root of the lung.

Course and Distribution of the Bronchial Arteries.—Each bronchial artery, when it reaches its bronchus, takes its course along it, lying at its posterior aspect, and, entering the lung, it divides and subdivides into branches, which accompany the bronchial tubes. The branches are small, and they lie close in contact with the outer wall of the tubes. Each branch of bronchial tube gets its branch of bronchial artery.

The vessels, as they pass along, give off branches, which run in the interlobular spaces, and supply the areolar tissue; they also give off branches, which are distributed to the coats of the blood-vessels, the nerves, and lymphatics.

If we examine the internal surface of the bronchial tubes, in a lung which has been well injected by the pulmonary veins or the bronchial artery, we find that there exists, in connection with the mucous membrane, a very fine plexus of capillary blood-vessels, which can be traced throughout the whole extent of the tubes; where the injection has been made through the pulmonary veins, this plexus is more completely filled, especially towards the termination of the tubes, than when made through the bronchial artery; but in the latter instance, numerous vessels are seen to be filled, especially in the larger tubes, and their arrangement is readily observed. If, now, we further examine this *superficial plexus*, and, whilst under the dissecting microscope, remove with a fine needle the plexus with the thin membrane in which it lies, we may see that its vessels communicate with other vessels which are more deeply seated. These latter I will call, for the sake of distinction, the *deep plexus;*

they are found situated beneath and between the muscular fibres, and they take a circular direction round the tubes.

The *superficial plexus* of the bronchial tubes consists of a series of capillaries having a diameter varying from $\frac{1}{1500}$ to $\frac{1}{750}$ of an inch; they run for the most part in the longitudinal direction, taking, therefore, the line of the elastic fibres; they are connected with each other by means of cross branches, and the communications they thus establish are very frequent, so that a network of considerable regularity results.

This plexus is derived from the deeper set of vessels, which are seen, on dissection, to come towards the surface from the deep-seated parts. These latter vessels pass off from the trunk which accompanies the bronchial tube, and, after perforating the fibrous and muscular coats, reach the mucous membrane. The branches from which the plexus is immediately derived, are those which, after they have passed through the muscular coat, run between it and the elastic fibres; many of these, as they pass forwards, terminate somewhat suddenly by giving off a number of branches from their extremity, which pass in all directions, but for the most part longitudinally.

The *deep plexus* consists of vessels which are distributed to the muscular fibres, and the deeper portions of the tubes. The vessels which supply the muscles take a circular direction, for the most part, running beneath and between the fibres. They have a much greater diameter than the vessels of the mucous membrane.

It thus appears that there are two plexuses of vessels in connection with the bronchial tubes, one which belongs to the mucous membrane, the other to the deeper structures; but that they are both derived from the same source, and I believe they both terminate in a similar manner.

The bronchial arteries, as they accompany the bronchial tubes, give off branches which are distributed to the areolar tissue, and the various blood-vessels and lymphatics.

The pulmonary arteries get an abundant supply of blood, and a somewhat fine plexus is seen to be formed beneath their lining membrane. A plexus is also seen in connection with the pulmonary veins.

In the lungs of some of the lower mammalia, I have succeeded in injecting thoroughly through the bronchial arteries the blood-vessels accompanying the lymphatics, and a somewhat fine network is formed of these vessels on the surface of the lung, where are also seen the vessels of the sub-pleural areolar tissue, which are continuous with those situated in the interlobular spaces. In the human lung I have only occasionally, and that very partially, succeeded in injecting the blood-vessels of the lymphatics, through the bronchial arteries, but I have found them injected in preparations which have been injected through the pulmonary veins.

In the areolar tissue of the pleura a plexus of vessels of a somewhat fine character exists, and beneath these are seen the branches of the pulmonary veins. In the human lung I have only very rarely succeeded in injecting the vessels of the sub-pleural areolar tissue through the bronchial arteries; in some instances I have partially succeeded; and in injecting a lung which has been the subject of old standing pleuritic inflammation in spots, I have found the sub-pleural vessels injected at such spots. These vessels, however, although they cannot be easily injected through the bronchial arteries, may be easily filled through the pulmonary veins; they are continuous with those situated in the interlobular spaces, and they may be seen terminating in branches of the pulmonary veins.

I have mentioned that in injecting the bronchial arteries in the human lung I have not succeeded in injecting completely the vessels of the extreme bronchial tubes; the cause of this I shall endeavour to explain presently. With regard to the lungs of the lower mammalia—as the sheep, calf, and pig—I have experienced no difficulty whatever. When a bronchial artery of one of these animals is injected, the bronchial tubes are filled to their extremity, the fine plexus of the mucous membrane, however, being less completely filled than when the injection is thrown in by the pulmonary veins; the sub-pleural areolar tissue is also well injected, and the fluid finds its way into the pulmonary veins.

Having thus stated the results of my injections, it becomes an important question to consider in *what manner*, and *where*, the communication which undoubtedly exists between the pulmonary and bronchial vessels takes place.

If the statement I have made with reference to the bronchial veins be true, it is clear that those vessels do not return the blood supplied by the bronchial arteries, except so far as the latter vessels distribute it to the bronchi and the structures about the root of the lung; and the next point we have to examine is, whether any communication exist between the bronchial arteries and the pulmonary arteries. Do the former vessels pour their contents into the latter, which already contain venous blood, and therefore might be considered as fit receptacles for it? In all the best injections I have made of healthy lungs, whether of man or of the lower animals, in which the injection was introduced by a pulmonary artery, I have never found the bronchial tubes injected, except in those cases, as I have previously stated, where the injection has filled the pulmonary veins. I have mentioned that I have occasionally injected the branches of the pulmonary artery, through the bronchial artery—a result which would seem to indicate a communication between the two sets of vessels; I believe, however, that no direct communication exists, and that the result is produced by the fluid passing first into the pulmonary veins, and then through the pulmonary plexus, and thus into the pulmonary arteries.

Setting aside those cases of accidental extravasation, and of effusion of the injected fluid from the blood-vessels of the coats of the pulmonary arteries into the latter vessels, both of which accidents may take place, especially if the preparation be not quite fresh, I have found that when the bronchial artery was injected, the fluid *soon* returned by the pulmonary vein; when this was closed, and the injection continued for some time, and the piece examined as soon as the injection had set, I have found that the pulmonary veins were well filled, the pulmonary plexus partially so, and that the injection had found its way into the branches of the pulmonary artery.

From careful injection and examination of a considerable number of specimens, both of the human and other mammalian lungs, I have arrived at the conclusion that no direct communication exists between the bronchial vessels and pulmonary arteries.

We now pass on to consider the communication which undoubtedly exists between the bronchial vessels and the pulmonary veins. Some ingenious theories have been advanced with reference to this subject, which I shall have to examine hereafter. It is unquestionably more easy to inject the vessels of the small bronchial tubes through the pulmonary veins than through the bronchial arteries, and it is also quite possible to inject the tubes to a certain extent through those veins, without injecting the pulmonary plexus. On the other hand, the injection which is thrown in through the bronchial arteries rapidly and readily finds its way into the pulmonary veins. These facts seem to prove that the blood which is distributed by the bronchial arteries is poured into the pulmonary veins, and that the whole vascular system of the bronchial tubes communicates with the same veins. The question here arises, Do the pulmonary veins in any way supply the bronchial tubes? It has been asserted that the superficial plexus of the tubes is derived from the "air-cells," and that the blood, after ramifying over the mucous membrane, passes into radicle pulmonary veins. If it be so, it appears to me that we ought to be able to inject the plexus through the pulmonary artery, but this we are not able to do. Further, it has been stated that the bronchial arteries supply no part of the mucous membrane of the bronchial tubes; to this statement I cannot assent; I have several preparations in my possession, prepared by myself, which altogether negative the assertion.

The following, I believe, will be found to be true with reference to the distribution and termination of the bronchial arteries.

The bronchial arteries supply the whole of the structures of the bronchial tubes, the coats of the blood-vessels, the nerves and lymphatics, and the areolar tissue of the lungs, but not the air-sacs, which derive their nutrition from the pulmonary plexus.* These vessels (the bronchial arteries) form two plexuses in the air-tubes; one, which supplies the muscles and deeper structures, the other, the branches of which are very minute, the mucous membrane. The branches of these two plexuses, as well as the vessels which supply the other structures mentioned, all terminate in the pulmonary veins; and the reason why it is difficult to inject fully the bronchial mucous membrane throughout the whole extent of the tubes, appears to me to be that the capillaries of the membrane are exceedingly small, whilst the capillaries in which the other vessels terminate are of a coarser character, and very readily allow the injected fluid to pass through them into the veins; and a route being *once* established, the fluid is diverted from its course to the mucous membrane, and only fills a part of the vessels of that membrane.

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* With regard to the nutrition of the air-sacs, no doubt can exist of the pulmonary vessels forming the source from which it is derived; no other vessels exist in the sacs, and in some animals the absence of bronchial arteries shows the capability of the pulmonary vessels for the function.

It may be said that the view which I have taken militates against the generally received opinion of the purity of the blood returned to the left side of the heart, for if the bronchial blood is poured into the pulmonary veins, it is returned to the left auricle without undergoing a process of aëration. I would answer, that the view is supported by anatomical fact, a basis on which all physiological theories should be founded.

That a distinct and free communication exists between the bronchial vessels and the pulmonary veins admits of ocular proof. I have seen, with the aid of the dissecting microscope, the small vessels passing from the outer surface of the bronchial tubes, and forming a small trunk, which terminated in a pulmonary vein.

THE AREOLAR TISSUE OF THE LUNGS.—This tissue exists in but small quantity in the lungs of man. It is found investing the various tubes, vessels, &c., which ramify in the substance of the lungs, and surrounding the different lobules. At the surface of the organ it is continuous with the sub-pleural areolar tissue. It is most abundant proportionately in the lungs of infants, and least so in old age. It usually contains, except in very early life, a quantity of pigmental deposit, which varies much in different individuals, but, as a rule, increases with increasing age. My observations on the fœtal lung tend to show that each pulmonary lobulette has its own separate sheath, and if so, it must be invested by a quantity of areolar tissue; this, however, is not discoverable in adult life. The areolar tissue enters the lungs at their roots, and accompanies their vessels and nerves. As I have before mentioned, the quantity is very small. It derives its nutrition from the branches of the bronchial arteries.

CHAPTER II.

PNEUMONIA—ITS MORBID ANATOMY AND PATHOLOGY.

(CLINICAL LECTURE.)

GENTLEMEN,—You have quite recently seen two cases of pneumonia under my care, and as you will constantly meet with cases of a similar character in our wards, I propose to occupy this and some following lectures in considering the nature of the disease, and the treatment which is most applicable to it.

There is no disease to which more attention has been directed, or which has been studied with greater care than pneumonia. From the high rate of mortality which has prevailed in it, no less than from the urgent symptoms it presents in its acute forms, it has ever had especial claims on the notice of the physician. The symptoms which characterise its onset, the phenomena attending its progress, the morbid changes by which it is accompanied, and the physical signs by which it may be recognised, have all been the subjects of careful consideration at the hands of the most able pathologists and practitioners of medicine. Nor has that which, after all, is the most important point in relation to the disease, its treatment, been wanting in an equal share of attention. Pneumonia may, indeed, be said to be the disease in which the various therapeutic systems for the cure of inflammation have been most largely tested. Blood-letting, tartar emetic in large doses or in small doses, mercury, opium, alcohol, the so-called expectant treatment, and the so-called restorative treatment, have severally had their advocates, and have been supported by statistics showing a greater or less mortality attending their use.

There can be no doubt that, of late years, important changes have taken place in this country in the practice of physicians in reference to this disease; that we no longer see the copious blood-lettings which were formerly practised, nor the administration of large doses of tartar emetic or mercury.

It is difficult to ascertain what is the average rate of mortality in the disease at the present day; and how far it differs from that which prevailed in past years. Statistics may in this respect be fallacious; the cases which have been grouped together by different observers may have differed materially in their nature, according as they have, or have not, included such as were complicated by some organic disease.

But still, looking at the mortality as it appears in the most valuable statistics which have been furnished us, we may, I think, safely conclude that the fatality of the disease has largely diminished since it was considered satisfactory to save three patients out of four attacked by it.

My own experience, both in hospital and private practice, induces me to believe that pneumonia, uncomplicated with any serious organic affection, such as Bright's disease or valvular disease of the heart, is by no means a fatal malady; that if patients suffering from it are seen tolerably early, and are judiciously treated, the mortality in it is low.

Into the wards of this hospital we receive a large proportion of cases of acute disease, and our patients consist very largely of seafaring men. I have thus had opportunities of seeing pneumonia, and other forms of acute inflammatory diseases, attacking those who were previously in apparently good health. Many of my patients have been sailors, doing their ordinary work on board ship, who, after exposure to cold, have been suddenly seized with symptoms of pneumonia, and have been brought to the hospital within a few days of the commencement of the attack. Some of these patients have presented all the general symptoms of high inflammatory fever, hot skin, full pulse, urgent dyspnœa, furred tongue, etc. They have, in fact, presented the symptoms which have been thought to indicate the necessity of general blood-letting. I think it important to allude to this, because I am sure that I occasionally meet with cases which present all the phenomena of high inflammatory fever, such as would have been largely bled some years ago; and, although it is quite possible that blood-letting would do such cases no harm, I know practically that they recover, and indeed rapidly, without the abstraction of a single drop.

I do not wish to enter into a consideration of the question as to whether there has been a change of type in diseases, which has modified our practice in this and other affections. My object is to discuss facts and results as they have occurred to me. I cannot, however, avoid remarking on the improbability of so remarkable a change having occurred in so short a space of time—a change which, if it is true with regard to man, must also be true with regard to some of the lower animals, as, for instance, the horse; for I believe I am right when I say that veterinary surgeons have more or less abandoned the practice of venesection, which used to form so important a feature in their practice.

You will find this question of the change of type in diseases, and of the asthenic form which inflammatory diseases are supposed by some to have assumed of late years, very ably discussed by Dr. Markham in the Gulstonian Lectures for 1864. I advise you to read those lectures. You will find, in the edition of them which has been lately published, a letter from Sir Thomas Watson, in which he expresses his concurrence in the views of Dr. Markham, and his conviction that the change of practice which has taken place of late years, the almost total abandonment of general blood-letting in acute inflammations, cannot be traced to any actual change of type in diseases, but to a modification in the opinions of physicians as to the nature of inflammation and the means by which it should be treated.

Before I proceed any further, let me direct your attention to certain points in connection with the morbid anatomy and pathology of pneumonia.

The conditions which characterise the different stages of pneumonic inflammation, engorgement, red hepatization, and grey hepatization, have been accurately described by various pathologists; and for the description of the ordinary appearances which these conditions present, I must refer you to your systematic works on medicine. Discrepancy of opinion, however, still exists with reference to some points connected with the morbid anatomy of the disease.

Such points are the following:

1. The blood-vessels which are involved in the inflammation.

2. The particular part of the pulmonary substance which is the seat of the disease.

Points of this kind can only be cleared up by a careful consideration of the anatomy of the healthy lung, and by a comparison of the latter with the organ when in a pneumonic state.

With regard to the blood-vessels involved in the inflammation, the opinions of pathologists are divided. Some believe that the capillaries of the pulmonary artery are the vessels essentially affected, whilst those of the bronchial arteries are also most probably involved. Some, as Dr. Morehead, consider that the bronchial capillaries are those mainly concerned; whilst others, as Grisolle, think that both sets of vessels are simultaneously affected, although, perhaps, in different degrees.

In considering this question, it is necessary to examine into the anatomical arrangement of the blood-vessels of the lungs; to define clearly the parts to which each set of vessels is distributed; and to ascertain the exact portions of the pulmonary substance which are involved in the pneumonic inflammation.

To refer briefly to the arrangement of "the ultimate pulmonary substance;" viz., that which constitutes the respiratory portion of the lung. Each terminal bronchial tube has connected with it a number of elongated cavities or "air-sacs." These are separated from each other by thin membranous walls, on which are found a number of cup-like depressions, alveoli, or air-cells. The series of air-sacs connected with the extremity of each bronchial twig, with its system of blood-vessels, &c., constitutes a *lobulette*. Each lobulette is perfect in itself, and has no lateral communication with adjoining lobulettes. A varying number of these lobulettes constitutes a lobule. Each lobule is, in the human lung, surrounded by a strong sheath, which possesses a good deal of elasticity, and is further connected with adjoining lobules by means of a small quantity of areolar tissue. The union of a number of lobules constitutes a lobe. (See Plates I. and II., figs. 1, 2, 3, 4.)

The pulmonary arteries are the only blood-vessels distributed to the respiratory portion of the lungs; viz., the walls of the air-sacs. These arteries, as soon as they reach the termination of the bronchial tubes, give off small branches, or arterioles, which take their course along the walls of the air-sacs, and break up into a capillary net-work, which constitutes the so-called pulmonary plexus. As these are the only vessels which are found in the walls of the air-sacs, they must be engaged, not simply in carrying blood for the special function of the lungs, but also for the nourishment of the tissue to which they are distributed. (See Plate III., fig. 7.)

Although the bronchial arteries pass along the bronchial tubes, and supply the structures of those tubes and the areolar tissue of the lungs, they yet send no branches to the walls of the air-sacs, which are solely occupied, as I have already stated, by the plexus derived from the pulmonary artery.

In speaking of the areolar tissue of the lungs, I wish it to be distinctly understood that no tissue of this kind is found in the walls of the air-sacs. These walls consist of a thin semi-transparent membrane, enclosing within it a quantity of elastic tissue together with the capillary plexus.

Areolar tissue is only demonstrable in the human lung, surrounding the bronchial tubes and the larger blood-vessels, and connecting the various lobules with each other. And although, as I have pointed out elsewhere, in the fœtal lung it is possible to separate each individual lobulette from those by which it is surrounded, no such separation can be made in after-life; and my opinion is, that any areolar tissue which may exist at birth around the lobulettes becomes subsequently absorbed. At all events, if any remain, the quantity is so small that it cannot be demonstrated.

Such being the distribution of the blood-vessels of the lungs, and the arrangement of the areolar tissue, the next point for consideration is, the exact seat of the pneumonic inflammation.

On examining, under the dissecting microscope, a piece of inflamed lung which has reached the stage of hepatization, it is at once seen that the seat of exudation is the air-sacs. These cavities are filled with solid matter; and, if the preparation have been kept in spirit for some time, moulds of the cavities can be drawn out. As the air-sacs are the seat of the exudation, it is obvious that the latter must be poured out from their walls. The structures composing these walls must, therefore, be the seat of the inflammatory process;

and as they contain no other vessels than those derived from the pulmonary artery, it is the branches of that vessel alone which are involved in the disease. (See Plate IV., fig. 8.)

In a piece of hepatized lung, exudation is sometimes found in the smallest bronchial tubes; at other times, it is absent from them, and merely fills the air-sacs, terminating abruptly at the spot where the structures forming the lobulette begin. The presence of this exudation in the bronchial tubes by no means proves that it has been poured out from their lining membrane; for it may have passed into the tubes from the air-sacs, in consequence of the over-distension of the latter.

In some cases of pneumonic inflammation, there is no reddening of the mucous membrane of the finest bronchial tubes—no *post mortem* appearances to show that there has been anything more than a simple uncomplicated inflammation of the air-sacs; whilst in other cases an increased vascularity of the bronchial membrane indicates the concurrent existence of bronchitic inflammation.

Some pathologists, in speaking of the morbid anatomy of pneumonia, have described the exudation as taking place in part into the "interstitial tissue." They have not, however, accurately described what they mean by "interstitial tissue;" and it is very important, in connection with this disease, that clear notions should exist in reference to this particular point. I have already mentioned that the lungs are not permeated throughout by areolar tissue; and that it only exists in certain parts, and in small quantities. The true lung-tissue—that which has been known as the parenchyma of the lung—consists of the walls of the air-sacs. These walls are firm and strong, but very thin. They consist of yellow elastic tissue, and a basement membrane inclosing the pulmonary plexus. No areolar tissue is found in these walls — a fact which a careful examination of the morbid appearances produced by pulmonary emphysema fully demonstrates; for in that affection the perforation of the lung-tissue which takes place produces a lateral communication between the air-sacs, but no extravasation of air into their walls.

Although, in pneumonia, the walls of the air-sacs swell and become somewhat thickened, chiefly in consequence, I believe, of the enlargement of the capillaries which they contain — partly, probably, from retaining some of the serous fluid which exudes from those vessels — they are yet not the seat of anything like extensive exudation. Their structure is, in fact, such as not to admit of it.

It is the opinion of at least one pathologist — Grisolle — that in pneumonia the capillaries are very probably augmented in number, as well as in size. It is impossible to speak positively with reference to this point; but my own opinion is decidedly opposed to that of Grisolle. The arrangement of the pulmonary plexus in health is such, that I believe no further development of vessels ever takes place. The branches of the plexus are very numerous, very closely set, and anastomose very freely with each other.

In the stage of grey hepatization, the air-sacs are still the seat of the exudation; and no destruction of their walls takes place, unless abscesses are formed. There is no interstitial suppuration. The exudation-matters, with which are mixed, but apparently not always, pus-corpuscles, and a considerable quantity of fat, are contained within the air-sacs, and, in the process of recovery, are either absorbed or expectorated.

From a consideration of the foregoing facts, I think it may be concluded, that pure pneumonia consists of an inflammation of the walls of the air-sacs; that the blood-vessels involved in the disease are the branches of the pulmonary artery which constitute the pulmonary plexus; and that the capillaries of the bronchial arteries are in nowise implicated, unless there is a concurrent bronchitis, which is an addition to, and not an essential part of, pneumonia.

If the opinions I have expressed with reference to the seat of pneumonia be correct, they tend to simplify our views of the nature of the disease. The affection becomes localised in the pulmonary plexus—a circumstance which gives it a greater importance than it would possess, did it depend simply upon a morbid state of the bronchial arteries, which are still held by some to be the nutrient vessels of the air-sacs, and, as such, the sole vessels implicated in pneumonia.

There are some circumstances which seem to me, apart from the anatomical considerations I have referred to, to bear strongly in favour of the views I have expressed. Amongst these, I would mention the severity of the fever which accompanies pneumonia, assimilating it, in this respect, very much to a blood-disease; and, again, the rapidity with which consolidation of the lung takes place. It is easy to account for this rapid consolidation, supposing the materials to be poured out from the great pulmonary plexus; but it is very difficult to understand how the small bronchial arteries could, thus rapidly, give rise to the effusion of so large a quantity of material, supposing them to be solely concerned in its production.

It has been objected to the view that pneumonic exudation takes place solely into the air-sacs, that, in certain forms of the disease, there is no expectoration: further, that *post mortem* examination shows that, in such cases, exudation has taken place into the "interlobular tissue." I have already explained my views with reference to the non-existence of any tissue around the air-sacs which could be the seat of exudation; and that, in the many specimens of pneumonic lung which I have examined, I have always found that the effused matters have been poured into the air-sacs.

With regard to the absence of expectoration in certain cases of pneumonia, and the inference that has been drawn from the circumstance —that the exudation is not poured into the air-sacs, I cannot think that the fact affords any such proof. Expectoration in pneumonia is a symptom which varies very much; it is often small in quantity when the inflammation is extensive, and, in fact, it bears no proportion to the amount of lung involved; nor can its entire absence throughout a case be admitted as proof that the exuded matters have not been poured into their usual seat. *Post-mortem* examination alone can afford proof of this; and, as I have before remarked, I have never found, in the many specimens I have examined, the exudation occupying any other site than that which I have referred to.

It is quite possible that the areolar tissue surrounding the lobules of the lung *may* be the seat of inflammation, and I have occasionally found this tissue thickened and indurated in old standing cases of lung disease; but an affection of this kind bears no relation to ordinary pneumonia even if it ever exist in an acute form.

CHAPTER III.

PNEUMONIA—ITS FIRST MORBID CONDITION AND EARLIEST PHYSICAL SIGNS—CAUSE OF THE CREPITATING RÂLE, ETC.

(CLINICAL LECTURE.)

GENTLEMEN,—Before I proceed to speak of the treatment of pneumonia, I wish to say a few words in reference to the early physical signs of the disease, and the morbid conditions by which they are produced.

The general symptoms and signs, which characterize the onset and progress of pneumonia, are so well described in your various systematic works, and will be so frequently illustrated by the cases which I shall have to detail, that I shall purposely abstain from any regular description of them here. I must, however, refer at some length to a phenomenon which I have noticed, and about the existence of which there is some difference of opinion.

I mentioned in the last lecture, that I am of opinion that engorgement is not the earliest morbid condition of pneumonia; and I also believe that crepitation is not the earliest physical sign of the disease. Crepitation is the auscultatory sign which characterizes the stage of engorgement, and, practically, is the first sign on which you can depend as indicating the existence of pneumonia. I shall have to speak of it again, and point out to you that it may, however, be heard when pneumonia is not present.

But, of the earliest morbid condition; I agree with the conclusions arrived at by Dr. Stokes, that there is a stage prior to that of engorgement, characterized by dryness, intense arterial injection, and, consequently, a bright vermilion colour, of the pulmonary membrane. In proof of the probability of this condition, I must appeal to the facts furnished by auscultation, viz., the existence of a harsh, loud, puerile respiratory murmur, preceding the crepitating *râle*.

It is very rarely that an opportunity is afforded us of making an examination of the chest in incipient pneumonia; and to this fact we must, I think, attribute the differences of opinion which have been expressed as to the earliest physical signs of the disease.

I have had two cases under my care in this hospital, in which I noted the existence of a loud, harsh respiratory murmur as an initial physical sign of pneumonia. In both cases there was acute primary pneumonia occurring in lungs previously healthy. I think it is important to note this; for, to render the observation of this particular phenomenon perfectly trustworthy, it ought to be made on a case, not where there is progressive inflammation, nor yet where there are consecutive attacks of inflammation, for the cause of the phenomenon might, under such circumstances, admit of some doubt; but where, the lung being in a healthy condition, inflammation of the organ comes on suddenly. Let me refer you to the following cases:—

CASE 1.—P. F., a carter, was admitted into the hospital, under my care, on August 8th, 1864. On the day of admission, at an early hour, he was out in a shower of rain, was very wet, and did not change his clothes. In the course of two or three hours he felt pains about the limbs, and had severe rigors.

When admitted into the hospital about mid-day, he was seen by the house-surgeon. He then complained of pain in the lower part of the left side. There were no febrile symptoms, and no abnormal physical signs about the chest.

On the following day, about noon, his condition was as follows:—The pulse was 120, and full; respirations 32; skin very hot and dry; tongue coated with a white fur. The pain in the left side had increased. There was no cough, but much dyspnœa. The percussion-sound and movement of the left side of the chest were natural. *At the lower and back part of the left lung a loud, harsh, peculiar respiratory murmur was audible.* No such sound could be heard elsewhere. The patient was ordered a grain of opium three times a day, with small doses of tartar emetic.

The next day the pain in the side was almost gone. The pulse was 104; the respirations were 28. The physical signs were as follows:—Deficient movement of the left side, dulness at the left base, with crepitating *râle* over the lower half of the left lung. The crepitating *râle*, which was distinctly of a pneumonic character, occupied, in fact, this day, the seat of the harsh, loud respiration of the preceding day.

It is needless to follow the history of the case further. The crepitation was succeeded by bronchial breathing and all the symptoms of confirmed pneumonia. The patient made a satisfactory recovery, and was convalescent on the eighth day of the attack.

Case 2.—D. M., a Frenchman, was admitted into the hospital, under my care, on January 23rd, 1865. Two days before admission, he was perfectly well. He complained of dyspnœa and pain in the chest. On examination, *a loud harsh respiratory murmur* was heard over the lower and back part of the left lung. The movements of the side were good, and there was no dulness. The breath-sounds over the opposite lung were normal. On the following day, the physical signs were as follows:—Slight dulness at the base of the left lung, and well-marked crepitation over about the lower half of the same lung. In fact, as in the preceding case, the loud respiration of one day was replaced by the crepitating *râle* on the next. The patient subsequently had all the symptoms of confirmed pneumonia—dulness, bronchial breathing, and rust-coloured sputa. He was convalescent about the tenth day.

From the observation of these cases, I cannot entertain the slightest doubt that, neither is the crepitating *râle* the earliest physical sign of pneumonia, nor engorgement its first morbid condition. It is true that I have never been able to demonstrate, by a *post-mortem* examination, the dryness of the pulmonary membrane and the arterial injection, which I believe to exist prior to the stage of engorgement; nor, indeed, would it, I think, be easy to satisfy the minds of those who are sceptical on the subject by any such examination; for they might consider the appearances the result of mere congestion. At the same time, this absence of *post-mortem* proof must not blind us to the facts which clinical experience teaches us.

As I have already mentioned, there is much difference of opinion as to the existence of this phenomenon; but before I speak of the objections which have been brought forward against the possibility of its occurrence, I wish to explain the way in which, I believe, this harsh respiration is produced, and to point out to you the condition in which I suppose the pulmonary membrane to be; and I shall take this opportunity of explaining to you what I consider to be—First, the cause of the ordinary respiratory murmur; and Secondly, the cause of the crepitating *râle*.

First, as to the respiratory murmur:—Various causes have been, from time to time, assigned for its production; and although, in a practical point of view, its exact seat and proximate cause may appear unimportant, provided we are familiar with the sound itself, and can rightly interpret the modifications of it which result from disease, yet it must be confessed that clear views of the physical phenomena of all healthy organic actions are very desirable; and, just as our knowledge of the simple manner in which the sounds of the heart are produced, has facilitated our diagnosis of cardiac diseases, so, more precise information than that we already possess, with regard to other points of a similar nature, cannot fail to be followed by beneficial results.

To the physical condition of the lung it is obvious that we must look for an explanation of the cause of the respiratory murmur; and there is one anatomical point, either unknown to those who have given their attention to this subject, or overlooked by them, which appears to me to offer a satisfactory solution of the phenomenon.

Without attempting to examine critically the opinions of others, I must content myself with observing that I believe the air-sacs of the lungs to be the seat of the murmur; and I shall now proceed to point out the arrangement which exists at the mouth of each air-sac, to which arrangement I am of opinion that the sound is due.

I have pointed out elsewhere the manner in which each bronchial tube terminates in a series of air-sacs; and the passage which has the most important bearing on the question of the cause of the respiratory murmur is the following:

"The air-sacs consist of somewhat elongated cavities, which communicate with a bronchial ramification by a circular opening, which is usually smaller than the cavity to which it leads, and has sometimes the appearance of a circular hole in a diaphragm, or as if it had been punched out of a membrane which had closed the entrance to the sac." (See Plate IV., figs. 9 and 10.)

This arrangement is best seen in the lungs of children and of adults. In old age it has frequently disappeared, more or less. It may be often well seen in a piece of lung, the blood-vessels of which have been injected with coloured size, and which, after being dried, has been subsequently soaked in spirit. By careful dissection under a microscope, the membrane, guarding the mouth of the sac, and narrowing the entrance to the cavity, is easily demonstrated. The membrane forms a part of the aërating walls of the air-sac, and has branches of the pulmonary artery ramifying in it.

It is obvious that a condition of this kind must have an influence on the passage of the air into the air-sac; that, to a certain extent, it must produce an impediment to the current of air, and thus give rise to a sound.

As the air is moved along the bronchial tubes it meets with no obstruction to its passage; but at the commencement of the air-sacs an opening exists which is smaller than the cavities between which it is placed. As the air-sacs expand with each inspiration, air must pass through the constricted opening. I believe that, in the passage of the air through this opening, the main element of the respiratory murmur consists.

The following facts appear to me to afford arguments in favour of the view I have advanced: the respiratory murmur is loud and well marked in infancy and childhood; it becomes modified in adult age, and in old age it is frequently very feeble. In the infant the membrane placed at the mouth of the air-sac is well-marked and uninjured; the opening in it has a clearly defined and sharp margin; and, moreover, it is smaller—not only absolutely, but I believe also relatively—than in after-life. In the adult, the air-sacs have undergone enlargement, and the membrane at their entrance is more or less perfect, according as the lung is in a more or less healthy state; whilst in old age the membrane has often, to a great extent, disappeared, apparently as the result of the wasting and absorption, which so frequently occur in the lungs of those advanced in life.

Further, the changes which take place in the character of the respiratory murmur in emphysema of the lungs afford an additional argument in support of this view. In this disease, in consequence of distension, rupture, and absorption, the air-sacs become much altered in character, and the membrane guarding the entrance to them entirely disappears as the disease progresses. The obstacle to the passage of air is therefore removed; and hence one reason of the extremely feeble respiratory murmur which characterizes the affection.

And now let me explain to you the way in which, I believe, this healthy respiratory murmur passes, first of all, into the harsh puerile respiration of incipient pneumonia, and subsequently into the crepitating *râle*, when the disease is fully established. It appears to me that the first phenomenon, which is merely an exaggeration of the healthy sound, is the result of the dry and swollen condition of the pulmonary membrane; that this gives rise to a constriction of the mouths of the air-sacs, and approximates them, therefore, to the condition which they present in childhood, when a loud respiratory murmur is usually heard. I see no reason to doubt that there is a dry stage in pneumonia, as well as in inflammation of mucous membranes. It is said that every stage of inflammation of serous membranes is marked by exudation; and it has, therefore, been inferred that such must be the case in pneumonia. But, although the lining membrane of the air-sacs resembles to a certain extent a serous membrane, yet it does not possess all the characters of such membrane. It consists, as I have already mentioned, of some yellow elastic fibres, a very delicate basement membrane covering the blood-vessels, and a layer of epithelium having somewhat the character of the epithelial cells found on serous membranes, but being by no means identical with them.

It has been objected to the view that there is a puerile respiration preceding the crepitating *râle* in pneumonia, that the sound which is thus described is nothing more than the result of a supplementary movement in parts around a spreading obstruction; that when this sound is heard, and is followed by crepitation, there has been, at the time when it was heard, consolidation of the lung in adjacent, more deeply seated portions. I think that the circumstances under which the sound was heard in both my cases negative the possibility of such an explanation of it. Take the first case. The patient was admitted at noon on the 18th of August, having been wet early in the morning, previously being in good health. He was carefully examined, and nothing abnormal was found about the chest; nor was there any fever. It will scarcely be inferred that pneumonia was present at that time. Twenty-four hours afterwards he was again examined. There was a good deal of fever; the respiration was hurried; and there was pain in the chest. There was no dulness; but a harsh respiration was heard over the back of the left lung. Now, is it at all probable that, during the short period that had elapsed since the man's attack, consolidation of the lung could have occurred — especially taking into consideration the subsequent progress of the case? For, after the lapse of twenty-four hours more, we had the stage of engorgement established in the more superficial portions of the lung, but no consolidation. I need not refer to the second case, for it presents features similar to those of the first.

I feel convinced that, in the two cases which I have detailed to you, this harsh respiration was an initial symptom of pneumonia; and, although it may not be a constant precursor of the crepitating *râle,* I believe it would be much more frequently met with, if we had more opportunities of auscultating our pneumonic patients in the early stages of their disease.

But now, as to the manner in which the crepitating *râle* is produced: I believe that its seat is in the air-sacs, and that it is caused by their expansion at the time when their walls are covered with the secretion which is poured out upon them. The expansion of the sacs, when they are partially filled with fluid, appears to me to afford the conditions necessary for the production of the *râle*. That it has its seat in the finest bronchial tubes, I cannot admit; for in some cases these tubes are found after death free from exudation.

There are conditions under which the crepitating *râle* may be heard when no pneumonia is present. In certain cases of œdema of the lung, I have heard a crepitation as pure as anything I have ever heard in the most typical pneumonia; and trusting, therefore, to this sign alone, you might in some cases be misled as to the nature of the disease; but, generally speaking, there is no difficulty. The ordinary symptoms of pneumonia are absent in these cases; there are dropsical effusions in various parts of the body, and other conditions which enable you to form a correct diagnosis. Still some cases are very puzzling, and, at first, are apt to mislead us; such, for instance, was the case of Scott, who died in L ward, and who, whilst in the hospital for valvular disease of the heart and dropsy, was seized with pneumonia. When I first heard the crepitating *râle* in this man, I thought it was the result of œdema of the lung; and it was only when other symptoms and signs developed themselves, that I became sure of the existence of pneumonia.

You may perhaps ask me how it happens that we hear the same sound in œdema of the lung as in pneumonia. The fact is, that the seat of exudation in the two diseases is the same; and in both conditions we have present, in the air-sacs, a certain amount of air and liquid exudation; the only difference being, that in one instance the liquid is somewhat more viscid than in the other.

CHAPTER IV.

PNEUMONIA—TREATMENT.

(CLINICAL LECTURE.)

GENTLEMEN,—I propose, in this and some following lectures, to speak to you of the treatment of pneumonia; and, in doing so, I shall refer you to cases which you have seen in the wards lately, and to others which have occurred in previous sessions, and of which notes have been taken. A careful record has been kept of all the cases of pneumonia which I have had under my treatment in this hospital. The symptoms and physical signs which have existed in each case have been observed at the bedside from day to day, and at once recorded, together with the treatment which has been adopted. It is only by a plan of this kind that you can arrive at anything like accuracy in reference to your cases, or that they can become valuable as standards of reference, or as illustrations of any method of treatment. In hospital practice alone is it possible to follow out such a system—to take down the details of cases of acute disease, so as to render them of real value in regard to any scientific inquiry. And I must here tender my thanks to the gentlemen who, for several years past, have filled the office of junior house-surgeon in the hospital, and to those who have acted as my clinical clerks, to all of whom I am largely indebted for the careful record that has been kept of my cases.

I may at once remark, that no single line of practice will, in my opinion, ever be found applicable to all cases of pneumonia. In the treatment of each case you must have regard, not simply to the amount of lung involved, or to the stage which the disease has reached, but also, and more especially, to the constitutional condition of the patient, the frequency and character of the pulse, and the antecedent circumstances, as far as you are able to ascertain them. Bear in mind that it is the patient himself, and not simply his diseased lung, which is the subject of your treatment.

You will have observed that the treatment which I adopt is not characterized by the exhibition of large doses of any of the so-called antiphlogistic remedies; that I never withhold nourishment from the patients when they can take it; and that I do not resort to powerful purgatives. On the other hand, you must have noticed that, in many cases, I prescribe stimulants at an early period of the disease, and that they often form the main therapeutic agent on which I rely.

Let me say a few words with reference to blood-letting. In the series of cases which I have tabulated, to be referred to in a future lecture, I have never resorted to venesection, and only occasionally, and that in the earlier numbers of the series, to cupping or leeching. I believe there are few cases now met with which are benefited by general bleeding; and that the abstraction of blood by the cupping-glasses or leeches is not often necessary. No doubt this local bleeding sometimes gives great relief to pain; but I think we can generally afford equal relief by the use of other measures which I shall allude to hereafter.

The natural history of pneumonia has been better studied of late years than it was formerly; and the tendency of the disease, when uncomplicated, to terminate favourably, has been brought out in strong relief. But, although it is highly probable that, a large number of cases of pneumonia would end in recovery if left to themselves—that is, if the patients were merely confined to bed and properly dieted—yet, there cannot be the slightest doubt that, even in such cases, the duration of the disease may be shortened, and the convalescence from it hastened, by the judicious use of therapeutic measures. We frequently meet with cases, in which, in consequence of neglect, the lung has remained consolidated for a considerable time—a condition which might have been rapidly got rid of by appropriate treatment.

I must tell you that I do not believe that we possess any single remedy which is specially, and specifically, curative of pneumonia; but, at the same time, I believe that there are certain agents, which, by their general effects on the system, exert a decided influence in the disease. An agent which has been much vaunted, and very largely used in pneumonia, is tartar emetic. It is a remedy which you have seen me prescribe occasionally; and, in some cases, it appears to me to be valuable. I shall refer to cases in which I fairly tried the effects of stimulants on the one hand, and of small doses of tartar emetic on the other, and in which the former proved decidedly injurious, whilst the latter soon gave relief to the symptoms. There are many cases, however, in which antimony must not be given at all—cases which are marked from the onset by a weak and rapid pulse, and in which it is plainly manifest that there is a want of constitutional power. Again, with regard to this remedy, there are few cases, I believe, which require, or are benefited by, a prolonged administration of it: nay, I think I may say that there are few cases that are not injured by such a practice; and whenever the drug produces a depressing effect, whenever it gives rise to sickness or purging, I believe it does harm, and should not be persevered with. It seems sometimes to promote perspiration, and thus to give great relief; further, it appears to have a beneficial influence on the pulmonary membrane, in rendering its secretion less viscid, and in facilitating expectoration.

It is rarely, if ever, necessary to give it in large doses. From one-sixteenth to one-fourth of a grain I have usually found quite enough. At the same time, there can be no doubt that larger doses—one to two grains—may be safely given in some cases, and continued even for days, without producing any of the so-called physiological effects of the drug—purging, vomiting, &c.; and in such instances the antimony appears to do good; the symptoms of the pneumonia yield during its administration, and convalescence becomes established. But, whilst these full doses of tartar emetic are well borne by some patients, I doubt much whether it is ever desirable to exhibit them; for I believe that we can get all the good effects which the drug is capable of affording by using it in smaller quantities. Of late I have treated the cases which I thought would be benefited by antimony with small doses of the remedy; but, in some of the earlier cases referred to in the table, half-grain, and grain doses were given.

The propriety or impropriety of administering alcohol in pneumonia is one of the most important questions in connexion with the treatment of the disease. Here, again, we find that no fixed nor definite rule can be laid down. Whether stimulants shall be given in large quantities or in small quantities, or be withholden altogether, must be decided from the general features of each case, and not simply from the fact that pneumonia exists. There can be no doubt, that many cases of pneumonia may be conducted to a satisfactory issue, without the administration of a single drop of alcoholic stimulants; further, that there are cases in which alcohol aggravates the symptoms, increases the distress, and retards convalescence. At the same time, there exist other cases which are as decidedly benefited by stimulants, and in which they should form the main therapeutic agent to be relied on. I shall endeavour hereafter to point out the symptoms which, in my opinion, indicate the necessity for the free administration of alcohol. I may here remark, however, that in a large proportion of the cases which have come under my care, both in hospital and private practice, I have given, apparently with decided advantage, a larger or smaller quantity of some form of alcohol, either wine or brandy, in the earlier stages of the disease.

The administration of calomel and opium, which used to form so important a feature in the treatment of pneumonia, and which received the sanction of some of the most eminent practitioners of medicine, has of late years fallen into disuse. There can, I think, be no doubt, that too high a value was placed on mercury as a remedy in the stage of hepatization, for which it was considered peculiarly applicable; and that it possesses no special properties for promoting absorption of the effused matters. As a purgative, mercury is very useful, as it tends to relieve the portal system, often overloaded in pneumonic inflammation; but, if given in frequent doses, or with the view of producing salivation, I believe that its effects, except in a few exceptional cases, will generally be more or less prejudicial. I have seen it given, and have had opportunities of watching its effects; but, in my own practice, I think I have not given it more than three or four times. In the series of cases referred to in the table, it was given once only; and my belief is that the progress of recovery was in nowise hastened by its administration.

The exhibition of opium is, I think, very desirable in many cases. It often relieves pain, allays the distressing cough which sometimes exists, and procures sleep. I have found that the pain in the side which so frequently accompanies pneumonia, and for which I used formerly to prescribe cupping or leeches, may be generally relieved by the administration of a dose of opium.

There are some other remedies which are frequently given in inflammatory affections of the lungs, about which I should like to say a few words. Among these is ipecacuanha, which may, I think, be advantageously administered in some cases of pneumonia. It sometimes nauseates, and prevents the patient from taking nourishment, and in this respect may become prejudicial. In such cases it ought to be omitted. It is not a remedy you must trust to in any severe case of the disease; but, as an expectorant and diaphoretic, it may be occasionally of some service.

Carbonate of ammonia and chloric ether — spirits of chloroform of the *Pharmacopœia*—are substances which I frequently prescribe, in combination, in pneumonia. They are both stimulants, and, when given together, seem often to have a beneficial effect. When I think a case will not be improved by small doses of antimony, I generally prescribe ammonia and chloric ether, either with or without alcoholic stimulants.

With regard to the administration of salines, such as citrate of potash and acetate of ammonia, I do not, as a rule, prescribe them either in this, or in any other inflammatory affection. I think it doubtful whether the routine practice of constantly administering these substances in inflammations is a desirable one. There can, however, be no doubt that they are sometimes agreeable to the patient, and afford relief to the distressing thirst which is occasionally present. Further, by supplying water and certain constituents to the blood, they may promote the action of the skin, as well as of other excreting organs, and thus have a curative effect. At the same time, I would remark, in referring to the action of the skin, that it by no means follows that the existence of the hot, burning, dry skin, so frequently met with in pneumonia, necessarily indicates the use of salines, or of antimony, or of any other of the so-called diaphoretic medicines. This condition is sometimes rapidly relieved by the sole administration of some form of alcohol; and, in fact, wine or brandy will occasionally be found the best diaphoretic we can use.

The administration of nourishment forms an important element in the treatment of this, as of all other acute affections. In the early stages of a severe attack, there is but little desire for food; and there is a risk, if the mere feelings of the patient are alone consulted, that nourishment may be withholden too long. It is not desirable to starve a patient even during the acute stage of the disease; but small quantities of such nourishment as can be taken, excluding solid food, may be safely allowed. There is necessarily a great waste going on during the attack; and, unless this is to a certain extent supplied by food, there will follow great prostration, which will seriously endanger the patient's safety when the acute symptoms have subsided. For the most part, in the early stages, the quantity of food given may be safely left to the desire of the patient. As the case progresses and the appetite begins to improve, the diet should be more liberal; and you will find that, as soon as convalescence is established, solid food will be borne. In such cases as require a very early and free administration of alcohol, nutrients should be given liberally from the first. Beef-tea and milk are usually well borne; and, if the former be properly made, a good deal of nourishment may be introduced into the system by means of it.

Let me say a few words with regard to the practice of counter-irritation in the treatment of this disease. At the commencement of the attack, and in its early stages, turpentine fomentations, or mustard poultices followed by linseed meal poultices, seem to act beneficially; but later in the disease, when consolidation has taken place, blisters are, I think, of more value. I believe they produce a really curative effect on the diseased blood-vessels; that, in fact, they cause contraction of the capillaries by reflex action of the vaso-motor nerves. They appear also to be useful in promoting absorption of the effused matters. Large linseed meal poultices may be applied to the chest; but I do not think they are as valuable in this disease as in bronchitis, in which they often afford great relief.

CHAPTER V.

PNEUMONIA—TREATMENT—USE OF STIMULANTS—CASES.

(CLINICAL LECTURE.)

GENTLEMEN,—In my last lecture, I pointed out to you the modes of treatment which I think are most to be depended on in pneumonia; and I now wish to draw your attention to the details of some cases in which the principles I have referred to have been practically applied.

There is one circumstance to which I have not specially alluded, but which is of much importance in reference to the treatment of this affection—viz., the constitutional condition of the patients who are attacked by it. There can be no doubt that in many patients there has been some deterioration of health which has acted as the predisposing cause of the disease; but, at the same time, it is quite certain that cases occasionally occur in which there has been no previous ill-health. In such patients, exposure to wet or to cold, or to both, has been the exciting cause of the attack. Instances of this kind we sometimes meet with in our wards, in strong, active labourers or seamen, who are suddenly struck down by the disease. At the same time, I believe that in private practice you will very rarely meet with such cases. Most of those attacked with pneumonia, whom you will be called upon to treat in private practice, and especially the more wealthy of your patients, will be people not exposed to great hardships, nor to the inclemencies of the weather; and in these persons you will generally be able to trace some antecedent depressing circumstances, some deterioration of health, or some organic disease, which has predisposed to the pneumonic attack. I need not now impress on you the importance of inquiring into all these points when you come to consider the question of treatment, as I shall have to refer to them more particularly hereafter. Let me, however, here remark that in all cases, not only of pneumonia, but of other acute diseases, you should ascertain at the earliest possible period, by a careful examination, whether your patient is the subject of any organic disease of the kidneys, heart, or liver.

If there be one symptom more important than another in pneumonia, and which affords a safer guide to treatment than any other single phenomenon which the disease presents, it is, I think, the pulse. Had we some means of correctly estimating the character of the pulse, we should have less difficulty in judging of the appropriate remedies for the various inflammatory affections we meet with. It is not simply the frequency of the pulse that is important, but the indications which it affords of the power of the heart, and of the amount of arterial tension which exists—in fact, its character, as showing whether a stimulating line of treatment is required or not. To estimate aright this latter point is one of the most difficult problems in medicine, and can only result—with our present modes of investigation—either from unusual powers of discrimination or prolonged clinical experience. It is quite possible that the sphygmograph may become a valuable instrument in this respect, but our use of it has been so brief that, at present, we can speak with no certainty as to its value.

I dwell on the importance of the pulse, and I would also impress on you the importance of other symptoms in this disease, such as the respiration, the hot skin, the expression of the countenance, the voice, the condition of the nervous system; for, after all, when the question of treatment comes to be taken into consideration, it is these altered physiological conditions which mainly guide us. Our physical diagnosis tells us how much of one lung, or of both lungs, is involved in the disease; but it does not point out the line of treatment to be adopted. One patient, with a small portion of lung involved in pneumonia, may present more severe constitutional symptoms than another who has double the amount of tissue inflamed; and, as it is the patient you wish to cure, so your remedies must be addressed to the relief of the constitutional symptoms which are present. As a rule, it may perhaps be said that in pneumonia, when the pulse is below 100, the case is not a grave one, and will yield to treatment of a simple character; but when the pulse rises to 110, 120, or upwards, the case assumes a much more serious aspect. *The more frequent the pulse, as a rule, the greater is the need for stimulants;* and it is very remarkable to see the effect they produce on the pulse when it is abnormally frequent in some cases of pneumonia.

Let me call your attention to the following cases:—

M. J., a shoemaker, 28 years of age, was admitted into the hospital on the 1st of February, 1866. He walked into the hospital, but was unable to give any satisfactory history of his case; we learned, however, that he had been ill three or four days. When I saw him soon after noon, his pulse was 140, there was great dyspnœa, and he complained of severe pain in the side. The skin was very hot and dry; he had a short troublesome cough, and had expectorated some viscid sputum streaked with blood. We found, on examining his chest, that there was dulness on percussion, with crepitation over the lower half of both lungs behind. In front, the sounds were healthy. Here then we had a case of acute double pneumonia in its earlier stages, with a pulse unusually frequent—a pulse which, in my opinion, indicated great debility. I ordered the man to have a tablespoonful of brandy every two hours, and strong beef-tea. In addition, I gave him five grains of carbonate of ammonia, chloric ether and squills, every three hours. On the following day the pulse was 130; there was still great dyspnœa, and a very hot skin. The stimulants were continued; ten grains of Dover's powder were given at night, and a blister was ordered. On the 3rd of February, the dulness on percussion was more marked over the lower half of both lungs, especially over the right base, where there was absence of vocal vibration, but the general symptoms had improved, and the pulse had fallen to 88. On the 5th, the pulse was still 88, and a little blood had been expectorated. On the 6th, we found less dulness at the base of the lungs, and the pulse was only 68. In fact, convalescence was fairly established. He was ordered a chop, and the brandy was reduced to four ounces daily.

I need not give you any more details of the case. On the 14th of February, the percussion and breath-sounds were normal. The patient took quinine and iron, and was discharged, well, on the 3rd of March, having been kept in the house, that he might regain his strength, and be able to resume his work on leaving us.

Here is another case, in which stimulants were given early in the disease, with very satisfactory results.

Joseph D., 18 years of age, a sailor, was admitted into the hospital on the 24th of February, 1865. He had been ill for about five days, and attributed his illness to having caught cold a few days earlier, having been previously quite well. No treatment had been adopted. When admitted, he complained of pain in the left side; the pulse was 110; the respirations were 40, and the expectoration was viscid and rust-coloured. We found, on examination, dulness over the base of the left lung, with crepitation. I ordered a tablespoonful of brandy every two hours, with a mixture of carbonate of ammonia, chloric ether, and ipecacuan wine, and turpentine stupes were applied to the chest. On the 25th, the pulse was 108; the respirations were 50. There was no pain in the side. The dulness was more marked, and extended to the front. There was distinct tubular breathing, of a ringing character, over the left back. He was ordered to continue the stimulants, and a blister was applied. On the 26th, the pulse was 100; the respirations were 48. There was coarse crepitation at the base of the lung, otherwise the physical signs were unchanged. On the 28th, the pulse was 98, and coarse crepitation was extensively heard over the back of the left lung. On the 2nd of March, the pulse had fallen to 88. There was less dulness on percussion, and the general symptoms were much improved. On the 4th, the pulse was 80.

Up to this time no change had been made in the treatment. He had taken six ounces of brandy daily, and the ammonia mixture every three hours. The brandy was now reduced in quantity, the mixture was ordered to be taken three times a day only, and a chop diet was given. The patient gained strength rapidly, and, on making an examination of his chest on the 14th, a few days before his discharge, we found the percussion and breath-sounds quite normal. He left the hospital on the 17th of February.

Now let me draw your attention to another case, which is interesting in two particulars: first, as presenting a peculiar feature of the pulse; and secondly, as showing the beneficial effects of a stimulating treatment.

Robert R., 23 years of age, a sailor, was admitted into the hospital on October 18th, 1866. He said he had always enjoyed good health, but was of somewhat intemperate habits. Three days before admission, after exposure to cold, he had shivering and sickness, followed by pain in the right side.

On admission, the pulse was 112, and slightly intermittent. No marked abnormal sounds were heard on auscultation. He was ordered, by the house-surgeon, a mustard poultice, to be followed by a linseed-meal poultice; and four grains of carbonate of ammonia, with some chloric ether, in a saline draught, every four hours.

October 19th, 11 a.m.—He had passed a bad night. Pulse 120, intermittent; copious expectoration.

I did not see the patient till 3 p.m. on this day. The pulse had then fallen to 92, and was very intermittent. The expectoration was rust-coloured. There was a troublesome cough. There was bronchial breathing in the right axilla. The heart-sounds were normal. He was ordered to continue the ammonia, and to have a table-spoonful of brandy every hour and a half, with beef-tea, gruel, and milk.

On the 20th, the pulse was still intermittent; crepitation was heard over the back of the right lung. *The right cheek was much redder and hotter than the left.*

October 21st.—He had passed a better night. Pulse 104, more regular. He had been purged several times. He was ordered to have five grains of Dover's powder immediately.

October 22nd.—Pulse 84, very slightly intermittent.

October 23rd.—Pulse 72, regular. There was slight dulness with bronchophony at the back of the right lung. Crepitation was heard in front.

I need not follow out the details of the case. The patient steadily improved. He took the eight ounces of brandy daily till the 27th, when the quantity was diminished to six ounces. On the 25th, he was ordered quinine, which he took until discharged on November 9th.

The points of interest to which I wish to draw your attention in the case are—the intermittent pulse, and the beneficial effects of the stimulants which were administered. The intermittent pulse is rarely met with in pneumonia, apart from organic disease of the heart. In this man there was no valvular affection of the heart; and there was no reason to suppose that the muscular substance was the seat of disease. The intermission of the pulse was simply a functional disturbance, the result of want of power—of deficient innervation. It afforded me a strong indication for the line of treatment to be adopted. You will meet with this intermission of the pulse in various functional disorders—in stomach diseases, and functional disorders of the liver, etc.; but whenever you meet with it in acute inflammatory affections, I believe it is invariably an indication for the free use of stimulants. The quantity of stimulants given in this case, eight ounces—a third of a bottle—of brandy, daily, was not large; but, continued as it was, for eight days, after which only six ounces were given daily, it represented a considerable quantity of alcohol taken into the system. The results cannot be considered otherwise than satisfactory. The treatment was commenced early in the disease, and was directed to meet that condition of the patient, the most prominent feature in which was the feeble and intermittent pulse.

There is another circumstance mentioned in the notes of the case which is of some interest—the increased temperature of one cheek, the right, the side of the pneumonia. This symptom is one which I have frequently noticed, and it has been made the subject of special observation by Dr. Gubler, a Parisian physician. I do not know that it is of much importance, either with reference to the diagnosis or treatment of pneumonia. It is interesting as occurring on the side of the inflamed lung; but, where it exists, there are other more prominent symptoms of the disease, and in some cases of pneumonia it is absent.

But although I have brought under your notice some cases of pneumonia in which the pulse was frequent, and in which stimulants were administered with decided advantage, you must not conclude that every case of pneumonia with a quick pulse will be benefited by a similar kind of treatment. Here is an illustrative case:

Leopold A., 29 years of age, a Norwegian sailor, moderately stout, was admitted into the hospital on November 20th, 1863. On admission, he complained of pain in the chest and cough, and the physical signs of bronchitis were present. He was ordered turpentine fomentations to the chest, and stimulating expectorants, with six ounces of port wine daily, and beef-tea. No improvement followed; and, on the 22nd, symptoms of pneumonia set in.

On the 23rd, the skin was hot; the pulse was 132. There was marked crepitation at the base of the left lung, with dulness on percussion; the right lung being unaffected.

On the 24th, the pulse was 128, and strong. There was great anxiety of countenance, and much dyspnœa; the skin was very hot, and the sputum rust-coloured. Crepitation and dulness were found over the lower half of the left lung, with some crepitation over the base of the right.

Up to this time, no change had been made in the treatment prescribed, and the patient was evidently getting worse. The stimulating expectorants and the wine were stopped; and a fourth of a grain of tartarised antimony, with acetate of ammonia, was ordered every three hours. In the evening, a small quantity of wine was given in mistake; but none was afterwards given until convalescence was established.

On the 25th, the pulse was 104; the respirations were 46; the sputa were frothy and copious, but less rust-coloured. There was no crepitation over the right lung, but slight dulness was found. There was marked crepitation all over the left lung, with deficient expansion of the left side of the chest.

On the 26th, the pulse had fallen to 88, the respirations to 32. The sputa were frothy and copious. The antimony was ordered to be taken during the day only.

On the 27th, the pulse was 80; the respirations were 32.

On the 28th, they were respectively the same. The breathing was natural over the right lung, but slight dulness remained at the back of the left. The antimony was stopped; and ipecacuanha wine, with tincture of squills, ordered; together with four ounces of port wine and chop diet.

On the 29th, the pulse was 76; the respirations were 28.

On December 1st, the pulse was 90; the respirations were 24. There was some return of crepitation on the left side in front, with slight fever. The wine was omitted; it appeared to act prejudicially.

From this date, the case progressed satisfactorily. On the 8th, the pulse and respirations were normal; and on the 11th the patient was discharged, well.

Here was a frequent pulse; and yet stimulants only served to increase the urgency of the symptoms, which, however, yielded rapidly to the administration of antimony. Now let me refer you to another case.

D. M., a Frenchman, was admitted into the hospital on January 23rd, 1865. I have already alluded to the case in speaking of the early physical signs of pneumonia. The patient was a fireman on board a steamer just arrived from Rotterdam. The weather had been very cold, and the man had been exposed to great variations of temperature. He was perfectly well, he said, two days before he came to the hospital; but he had since felt pains about his chest, and had suffered from difficulty of breathing. He was of moderate height, of somewhat spare build, and fifty-five years of age. I saw him shortly after he was admitted. I found him with a hot and dry skin, flushed face, a furred tongue, a pulse of 100, full and strong, quick respiration, and occasional cough. On examining his chest, we found the movement and resonance good everywhere, and the breath-sounds normal, except over the lower and back part of the left lung, where there was a loud, harsh, respiratory murmur. No crepitation could be heard anywhere. I expressed an opinion that this was a case of pneumonia in the early stage, and that before long the harsh respiration heard over the left lung would be replaced by crepitation. I thought this would be a good opportunity for trying the effects of alcoholic stimulants in the onset of the disease. Nothing had been done for the patient before his admission. I accordingly, rather in the way of experiment than otherwise—for I was doubtful of any good result being produced—ordered a tablespoonful of brandy to be given every three hours, and turpentine stupes to be applied to the chest. This treatment was commenced about one o'clock p. m. I left directions with the house-surgeon that the brandy should not be discontinued, unless it appeared to do harm. The following are the notes of the progress of the case:—

9 p. m.—He appeared much worse; had great dyspnœa. Pulse 120, very full and strong, incompressible; respirations 40. He complained of a sense of oppression in the chest, and a feeling of suffocation; cheeks very flushed; great thirst. Crepitant rhonchus was heard at the left base. He was ordered to continue the brandy, and to take four grains of carbonate of ammonia, with twenty minims of spirits of chloroform, in camphor mixture, every four hours.

11 p. m.—He had had one dose of the mixture, and seemed still worse than at nine o'clock. The dyspnœa was so urgent that he could not lie down, and was obliged to be propped up in a bed-chair. Finding that the stimulating treatment was, to all appearances, doing the patient harm,

the house-surgeon very wisely stopped it, and ordered fifteen minims of antimonial wine, with the same quantity of ipecacuan wine, and a little sulphuric ether, with acetate of ammonia, to be given every three hours.

I wish you carefully to note the result of this change in the treatment.

On January 24th, at 2 p. m., he was much better. He said he began to improve after the second dose of the mixture. Pulse 92, less strong and full. Respirations 24. He had slight pain in the front of the chest, but the sensation of oppression was almost gone. The skin was less hot, and the face less flushed. The expectoration was frothy, not rust-coloured. There was diminished movement of the left side, and dulness of the lower part of the left lung, with abundant crepitant *râles*. He was ordered to continue the mixture and fomentations.

9 p. m.—Pulse 100. Respirations 30. He had been rather sick. He was ordered to take the mixture every four hours.

January 25th.—Pulse 100. Respirations 28. Skin moist. The mixture caused sickness. The expectoration was very tenacious and rust-coloured. The dulness at the left base was not so marked; crepitation abundant. He was ordered a tablespoonful of port wine every three hours, and to continue the mixture without the antimony.

I omitted the antimony on account of the sickness, and again tried the effects of stimulants in small quantities.

January 26th, 10 a. m.—Pulse 100. Respirations 26. He had no pain in the chest. The sickness had ceased. The expectoration was frothy and abundant. Crepitation was coarser than on the previous day. A blister was ordered to be applied to the left side, and some ipecacuan wine and sulphuric ether were given every four hours.

January 27th.—He had passed a bad night in consequence of cough and dyspnœa. The expectoration was very tenacious and rust-coloured. There was tubular breathing at the left base; less crepitation. *Rough and loud breathing was heard at the right base.* He was ordered by the house-surgeon a tablespoonful of brandy every two hours.

2 p. m.—Pulse 108. *Distinct crepitation was heard at the right base.* It was evident, from the symptoms which the patient presented when seen by me at two o'clock, that there was a return of the inflammation, and that the right lung was becoming involved. I felt convinced that the stimulating treatment was doing harm, and I accordingly stopped it altogether. I gave no more alcohol till convalescence was fairly established. He was ordered a sixth of a grain of antimony, with a few drops of laudanum, every two hours.

9 p. m.—He seemed much better. The cough was diminished, and the expectoration was more frothy. Pulse 104. Respirations 30. Abundant crepitation was heard on the right side.

January 28th.—Pulse 96. Respirations 24. He had passed an excellent night. He breathed quite easily, and had much less cough. The physical signs were improving. He was ordered to take the mixture every four hours.

January 29th.—Pulse 88. Respirations 24.

January 30th.—Pulse 88. Respirations 24. He was convalescent; and was ordered to take the mixture three times a day, and to have four ounces of port wine daily.

January 31st.—Pulse 84. Respirations 24. He had been rather sick; otherwise he was going on well. The mixture was stopped, and he was ordered six ounces of wine daily, and a chop for dinner.

February 1st.—Pulse 84. Respirations 20. The percussion-sound at the right base was almost natural. Dulness and tubular breathing were heard at the left base.

February 4th.—Percussion and respiration were normal on the right side. He was ordered quinine, four ounces of port wine, and two ounces of brandy, daily.

February 15th.—Respiration and percussion were normal on the left side.

February 22nd.—He was discharged, well.

I have referred at length to this case, because I think it is a very instructive one; especially when compared with other cases which I have already referred to, or which I shall hereafter refer to. It serves to show very forcibly that, as I said before, it is not every case of quick pulse which requires stimulants. When I tried the stimulants first, it was at the very onset of the disease, and there cannot be a doubt that they proved prejudicial; when I resorted to them the second time, the disease was subsiding, but two days of the treatment caused a relapse and a return of the unfavourable symptoms, which disappeared when the alcohol was omitted.

CHAPTER VI.

PNEUMONIA COMPLICATED WITH DELIRIUM—CASES—TREATMENT.

(CLINICAL LECTURE.)

GENTLEMEN,—There is one symptom which you will occasionally meet with in pneumonia, the nature of which you ought to be acquainted with—I mean delirium. You are aware that delirium occurs, not only in diseases of the brain and its membranes, but also in many specific fevers, and in some acute inflammations.

Setting aside the symptom when it arises from cerebral disease, let us briefly consider its pathology when it occurs in connexion with a specific fever, as for instance, scarlatina, or in the course of a local inflammation, as pneumonia, pericarditis, or erysipelas.

I must tell you that delirium used to be considered, and indeed, not very long ago, as a symptom essentially connected with inflammation of the brain or its membranes, and this view led to a treatment, the object of which was to subdue the inflammation that was supposed to exist. *Post-mortem* examination, however, revealed the fact that in many cases in which delirium had occurred, no results of inflammation within the cranium, could be discovered. Indeed, we now know that acute inflammation of the brain or its membranes is a very rare disease in adults, and that, in the large majority of cases in which we meet with delirium, there is no structural change of an inflammatory kind going on within the cranium.

We have abundant evidence that a poisoned condition of the blood—or an altered state of the blood from the circulation in it of some foreign substance—is alone sufficient to cause delirium. Take, for instance, the effects produced by the imbibition of alcohol, or by the inhalation of chloroform. When death takes place from an attack of delirium tremens, no structural alteration is discoverable in the brain. The delirium of this affection seems to be the result of the altered condition of the blood.

When, therefore, we meet with delirium in the course of a specific fever, as scarlatina, or in the progress of pneumonia, pericarditis, or erysipelas, may not the symptom be due to a like cause? Nay, has not clinical experience shown the strong probability that such is the case? And when an opportunity has been afforded of making a *post mortem* examination, have we not seen that not only has there been no congestion nor inflammation of the brain, but that occasionally the brain has even appeared to be in a somewhat anæmic state.

I believe these facts are becoming more and more recognised and acted on; but yet I doubt whether they have been sufficiently impressed on our minds, and whether they have yet led to the general adoption of a practice in accordance with the indications they afford; whether, in fact, we are not too apt to consider delirium, occurring in such cases as I have referred to, as the result of organic changes going on within the cranium, and to direct our remedial measures in accordance with this view.

That you may fully understand the view which I wish to impress on your minds, viz., that delirium is, in the main, of the same nature, whether it occur in a specific fever, as for instance scarlatina, or in pneumonia, or pericarditis, I must refer you to some illustrations.

I attended, a few years ago, a gentleman of middle age attacked with scarlatina. I saw him on the fourth day of the attack. The rash was not well out; the throat was sore; the pulse 120; and the fever rather high. The symptoms may be characterized as, at that time, severe, but not urgent. On the following day—the fifth of the disease—the general symptoms were much the same, except that there was great excitement in the patient's manner, with restlessness, flushed face, and injected conjunctivæ. Moreover, the urine was albuminous. On the following day—the sixth of the disease—I was told my patient had passed a bad night, and had been very restless and delirious. He was excited, talked in a rambling manner, but, when spoken to, answered sensibly. He complained of violent palpitation of the heart, which he said was a steam-engine working in his inside. I examined his chest, and found no evidence of inflammatory mischief. On the next day—the seventh of the disease—he had passed rather a better night, but still had been delirious. In the afternoon of that day, he began to talk in an incoherent manner; got up, crawled to the bottom of his bed, and was obliged to be put back by his attendants. When I saw him at night, he was more quiet, and the pulse was less frequent; but there was extreme prostration. From this time, no delirium occurred; and, with the exception of some slight diphtheritic symptoms, which manifested themselves on the eighth day, and a somewhat alarming prostration, the patient progressed satisfactorily to recovery.

Now, what were the measures which were adopted in this case, under which the delirium disappeared, and a favourable issue resulted? I must tell you that the delirium was of a severe type; it was not the mild delirium we so frequently meet with, but such as to require, for two or three days, constant watching, and, on more than one occasion, actual personal restraint.

On the day on which I saw the patient, he was taking chlorate of potash and steel, with small quantities of wine. On the following day, the steel was omitted, the wine and potash were increased, and free nutrition was ordered. As soon as the delirium occurred, notwithstanding the flushed state of the face, I considered the symptom to be due rather to a poisoned condition of the blood, and perhaps an anæmic state of the brain, than to any congestion or inflammation of the contents of the cranium; and, acting on this view, the only modification I made in the treatment was to increase the quantity of stimulants, and to insist more urgently on the free administration of nourishment. These measures were fully carried out, and the quantity of stimulants given was large. The chlorate of potash was continued up to the eighth day of the disease, when two grains of quinine were given every four hours.

I have referred to this case, because it is an instance of severe and alarming delirium occurring in the progress of a specific fever, not met by any abstraction of blood, nor by any remedy addressed to the head itself, but by free stimulation and nutrition; and with the most satisfactory results. To the early, persistent, and regular carrying out, night and day, of these measures, I attribute the patient's recovery; for, in spite of all the stimulants that were given, on the eighth and ninth days of the disease, when the fever-symptoms and delirium had subsided, the pulse became intermittent, and the patient lay for some time in a semi-syncopal condition, from which he was roused by a very liberal administration of brandy. I have mentioned that the urine was albuminous. On the fifth day, it was smoky, and gave a large deposit with heat and nitric acid. The alcohol did not seem in anywise prejudicial to this condition; on the contrary, during its administration, the smoky colour became daily less, the albumen diminished in quantity, and both conditions soon disappeared. No dropsical symptoms followed the attack.

Cases of this kind are frequently met with. I might relate others; but this one will, perhaps, be sufficient to illustrate the principle to which I have directed your attention. Had the delirium been dependent on inflammation or congestion of the brain, the treatment adopted might have endangered, or even destroyed, the patient's life. The result, I think, afforded a strong proof that no such condition existed.

But, to pass on to the delirium which we often meet with in the course of a local inflammation. The symptom comes on, for the most part, in patients who have indications of debility, and in whom the pulse is quick and feeble. It resembles, in my opinion, the delirium which is called traumatic.

An acute disease, like an accident, interferes with the usual habits of life, and when there has been an over-indulgence in alcoholic liquids, severe mental exhaustion, or anything which has led to impaired nutrition, it is very probable that results, similar in their character to those which often follow an accident, will arise in the progress of the acute affection, unless they are anticipated by the practitioner.

But there is, further, in these cases of inflammation, and more especially in some, as rheumatic pericarditis, an additional cause of the symptom in the unhealthy condition of the blood; this—together, perhaps, with the feeble action of the heart, consequent on its impaired nutrition, and often the mechanical interference with its movements—becomes the exciting cause of the delirium.

These remarks will also apply to the delirium which occurs in erysipelas. I have seen a large number of cases of this disease, and often as I have seen it attacking various parts of the body, I have never seen an instance in which there has been a metastasis to the brain or its membranes. Delirium I have seen very frequently, varying in degree, from the slightest possible disturbance of the normal function of the brain, to that which required constant watching; but I have never seen actual inflammation within the cranium as revealed by the scalpel. And does not the treatment with which we meet these cases of erysipelas, even when delirium exists, bear out this view of the pathology of delirium? Do we not find, that the purely sustaining and stimulating treatment is that on which we can most safely rely, to carry out patients through a severe attack of the disease?

Now, if what I have told you be true, with reference to the nature of delirium, you have very plain indications for its treatment. Your object must be to improve the nutrition of the brain, and to give vigour to the circulation; and as you accomplish these results, you will find that the delirium will disappear. You must not be deterred from exhibiting stimulants from any fear of the existence of inflammation of the brain, nor must you surround your patient's head with leeches under a similar dread. The delirium partakes of the nature of delirium tremens, and requires to be met with remedies such as you would prescribe in that affection.

In all your cases of pneumonia you should be on the watch for an outbreak of this symptom, and you should endeavour to check it in its early stages. Want of sleep, restlessness, slight staring of the eyes, and a slight tremor of the hands, are indications of approaching delirium. The administration of a few doses of opium, and the exhibition of stimulants and nourishment, on the appearance of any of these symptoms, may at once arrest the attack. Let me refer you to the following cases, in which delirium occurred, and was, I believe, cut short by the treatment adopted.

Thomas R., aged 21, a hawker, was admitted into the hospital on December 28th, 1861. The patient stated that he was quite well on December 24th, and was out hawking on that day. On the morning of the 25th, he was suffering so much from pain in the side, that he was unable to leave his bed, which he kept until he was brought to the hospital. Six leeches had been applied to the left side the night before his admission. He said he had been drinking "hard" for about a fortnight before he was taken ill.

When admitted, he complained of pain in the left side, and shortness of breath. The pulse was 116, and hard; the respirations were 60 per minute. Both lungs were resonant in front, with loud breath-sounds, especially on the right side. The right lung was resonant behind, and there was fine crepitation at the extreme base; elsewhere, the breathing was good. There was loud bronchial breathing, with fine crepitation, over the whole of the back of the left lung, with dulness below the level of the spine of the scapula and towards the axilla. The sputa were tenacious and rust-coloured. The urine was free from albumen. He was ordered a purgative of calomel and colocynth, with a quarter of a grain of antimony in a saline draught every three hours, and beef-tea for diet.

At 9 p.m., he was breathing very rapidly, the pulse was 110, and strong. He was ordered to take the draught every two hours.

On the following day (the 29th), he said he felt better, but had not slept. He complained of pain in the side on coughing. The bowels had acted; there had been no sickness; and he was perspiring freely. He had had slight epistaxis. The pulse had fallen to 74, and was softer; the respirations were 28. The right lung was free from crepitation. On the left side the crepitation was less distinct; but the bronchial breathing was more marked; and there was distinct bronchophony. The antimony was ordered to be discontinued during the night.

On the 30th, he had slept but little, had been slightly delirious during the night, was restless, and had an anxious expression of countenance. Pulse 78; respirations 40. The dulness was more marked at the back of the left lung. The sputa were less discoloured. He was ordered half a drachm of laudanum at once—the dose to be repeated in four hours, unless he slept; to have two ounces of brandy at night; and to take the draught every four hours, with the addition of ten minims of laudanum. He took the two doses of laudanum, and slept.

On the next day, he was better. Physical signs about the same. He was ordered a blister, and three ounces of brandy.

On the 1st of January, he had slept well. Pulse 72; respirations 20. The mixture to be taken every six hours. To have two extra ounces of brandy at night, strong beef-tea, etc.

On the 2nd, the pulse was 64; respirations 20. There was dulness on the left side below the spine of the scapula, with bronchial breathing, but less harsh than before. At the extreme base of the lung, the dulness was very marked, with very faint breath-sounds. The mixture was omitted, and a grain and a half of opium ordered every night, with four ounces of wine and two ounces of brandy daily, and chop diet.

On the 3rd, the pulse was 88; respirations 28. He was ordered six ounces of wine and an ounce and a half of brandy daily; and a grain of opium three times a day.

On the 5th, the brandy was omitted; on the 7th, the pulse was 68; respirations 15. The breathing was nearly natural over the back of the left lung, except at the extreme base.

On the 9th, the opium was omitted; on the 12th, herpes appeared over the trunk; otherwise he was well.

From this date he improved rapidly, under the influence of quinine and wine. He was discharged, well, on January 21st.

Now, let me draw your attention to another case of pneumonia, in which delirium occurred:—

Robert R., 43 years of age, a porter, was admitted into the hospital on the 1st of February, 1867. He had been well, he said, up to the 28th of January, viz., three days before admission, when he was attacked with shivering, cough, and pain in the chest. Nothing had been done for him, and he had been daily getting worse. He complained of cough, shortness of breath, and pain in the left side; the tongue was moist, and covered with a white fur; the bowels were loose; the pulse was 106. There was deficient movement of the left side of the chest, with dulness over the lower two thirds of the left lung behind. There was marked bronchial breathing over the middle of the same lung, with crepitation at the base. The front of the left, and the whole of the right lung, were free from morbid sounds. He was ordered a mixture of aromatic spirits of ammonia and chloric ether, mustard and linseed-meal poultices, four ounces of brandy, with beef-tea and milk for diet. On the following day (February 2nd), he had passed a good night. The pain in the side was relieved; the pulse was 104; and the respirations were 28. He had expectorated some rust-coloured sputa. There was tubular breathing over the lower three-fourths of the left lung, most marked at the middle and towards the axilla. There was a "nervous" expression about the patient and a restlessness of manner such as often precede an attack of deliruim. I ordered him to have a tablespoonful of brandy every two hours.

On the 3rd of February, I found that he had not passed a good night — that he had been restless, and slightly delirious. He was suffering a good deal from dyspnœa. The pulse was 104. The sputa were rust-coloured, and more copious than on the previous day. There was diminished area of dulness at the back of the left lung, and the tubular breathing was less marked. I increased the quantity of brandy to eight ounces instead of six, in the twenty-four hours; and I ordered a grain and a half of opium to be given at bed-time, if he were restless or wakeful.

On the 4th, he was decidedly better. He had passed a good night. He took the opium between nine and ten o'clock, and was afterwards free from restlessness and tendency to delirium. The pulse was 104. The physical signs were unchanged. A blister was ordered to the left side.

On the 5th, the pulse was 100; the respirations were 24. He had had slight delirium after midnight. He was ordered to have a grain of opium at bed-time. He slept well after taking the pill, and the following day he was much better. The pulse was down to 88. On the 8th the pulse was 73; the respirations were 22. The physical signs were improving. He was ordered a chop. On the 9th, the brandy was diminished to six ounces daily, and tincture of bark was added to the ammonia mixture. On the 12th, eight ounces of port wine were ordered instead of the brandy. On examining the chest on the 16th, we found the breathing almost natural over the back of the left lung. We kept the patient in the hospital till the 26th, in order that he might regain his strength.

There can be little doubt that in both these cases the severity of the attack of delirium was diminished by the early exhibition of opium and stimulants. In the first case the patient was of somewhat intemperate habits, and I watched carefully for the early symptoms of delirium, which I quite expected would supervene. For two days after his admission he had no sleep, although the pneumonic symptoms were relieved, and the pulse had fallen to 78. There was no decided delirium, but the restlessness and anxious expression of countenance, which were remarked on the 30th December, convinced me that unless more sleep were procured we should in all probability have the symptom developed. The administration of some laudanum procured sleep, and from that time the man began to improve, and with the aid of a moderate allowance of stimulants he progressed satisfactorily.

In the second case, the delirium was somewhat more marked than in the first; but it was checked, by the exhibition of brandy in half-ounce doses, every hour and a half, and opium at night. The man had a severe attack of pneumonia, although his pulse never became very frequent. He was treated from the first by stimulants, which were increased when the symptoms of delirium appeared.

The cases I have referred to will, I think, be sufficient to show you the mode of treatment, which I have found most satisfactory in pneumonia complicated with delirium. You may meet with instances in which the delirium is more violent than in those which I have mentioned, and you may have to give stimulants and opium more freely than they were given in those cases; but, by careful watching, you will generally be able to detect the early symptoms of the threatened attack, and, by timely use of such measures as I have recommended, to mitigate its severity, or perhaps, to ward it off altogether.

CHAPTER VII.

PNEUMONIA OCCURRING DURING RHEUMATIC FEVER.

(CLINICAL LECTURE.)

GENTLEMEN,—I wish to call your attention to-day to the subject of rheumatic pneumonia, or, in other words, pneumonia occurring during rheumatic fever.

Pneumonia is by no means infrequently met with in acute rheumatism. It forms, when it occurs, a serious complication, and sometimes, as in one of the instances I shall relate to you, it assumes a very severe type, and threatens the life of the patient. In such cases, it becomes the prominent feature of the rheumatic affection, and requires your special attention.

Let me say a few words as to its morbid anatomy. I do not believe that it possesses any peculiarity in this respect. Some physicians think that in rheumatic pneumonia the effusion is not poured into the air-sacs, which they admit are the chief seat of it, in ordinary pneumonic inflammation. They consider that the intervesicular areolar tissue is mainly the seat of the effusion. I hope I made it sufficiently clear to you, in a former lecture, that such a view is untenable, from the absence of any such tissue in the lungs. Again, it has been argued, because in some cases of rheumatic pneumonia there are no characteristic sputa, that the effused matters cannot be poured into the air-sacs. But this argument would equally apply to ordinary cases of pneumonia in which there is no expectoration. The fact, however, is, as I have mentioned before, that the absence of sputa is no proof whatever that the air-sacs are not the seat of the effusion. Further, although some cases of rheumatic pneumonia pass through their stages without giving rise to the characteristic expectoration, there are other cases in which it exists. I know of no feature which is peculiar to rheumatic pneumonia, or by which you can diagnose it, setting aside the affection of the joints with which it is associated. It corresponds in its symptoms, and in its progress, with ordinary pneumonia. The treatment which I have adopted in the affection is very similar to that which I have used in pericarditis. I have given, in some instances, carbonate of potash and opium, and a larger or smaller quantity of stimulants, according to the state of the patient. I am not aware that I have ever treated rheumatic pneumonia with antimony, and I am sure that I have never in such a case abstracted blood. A depressing agent like antimony can, I think, scarcely be desirable when there is acute rheumatism; and, in my opinion, the same remark will apply to mercury, which some physicians consider of especial value in rheumatic pneumonia.

All the cases of rheumatic pneumonia which have been under my care have terminated favourably; and it seems to me that the disease has a strong tendency to get well, provided the strength of the patient is not lowered by injudicious treatment. It may be necessary, as in one of the cases I shall relate, to give stimulants very freely, and you must not be deterred from giving them, under the impression that they will aggravate the rheumatic affection. You must be guided by the general condition of your patient, by the rapidity and strength of the pulse, in deciding as to the quantity of stimulants to be given, or whether they must be withholden altogether.

Some of you will recollect the following case, which occurred during the summer session:—

Thomas K., 16 years of age, a grocer's assistant, was admitted into the hospital on the 13th of June, 1867. He had just arrived from America, and had been laid up on board ship for about ten days. He told us that he had had typhus fever last year, that in March of the present year (1867), he had expectorated a small quantity of blood, and that in May he had brought up a large quantity. We found him suffering from acute rheumatism, involving the wrist and knee joints especially. He was in great pain, very weak, much emaciated, and quite unable to move. I ordered him ten grains of bicarbonate of potash, with a little laudanum, every four hours, and a grain of solid opium every six hours. I gave him beef-tea and milk for food.

On the following day, the pulse was 128. He was perspiring very profusely, and had had no sleep. You may recollect that I told you that the prominent feature we had to deal with was the extreme debility, and that, although there was an acute rheumatic affection of the joints, I considered it desirable to give stimulants. I therefore omitted the mixture, but continued the opium, and gave an ounce of port wine every three hours.

On the 15th, the pulse was 132. The tongue was moist and beginning to clean, and there was less pain; but the patient had not slept. We examined the urine, and found it free from albumen, and of sp. gr. 1.030. I increased the quantity of opium to a grain every four hours, and I ordered an ounce of wine to be given every two hours.

On the 16th, the pulse was 120. The bowels not having acted since his admission, he was ordered an aperient, and at night four additional ounces of wine were given by the house-surgeon, making sixteen ounces altogether during the day.

On the 17th, I found that he had passed a bad night; the pain in the joints was severe, and he had perspired profusely. The respiration was hurried, and there was some cough, but he was unable to expectorate from extreme weakness. He was lifted up and supported in the sitting posture, so that we might examine his chest behind, as I suspected some lung complication. We had examined the front of the chest before, and had discovered some bronchitic *râles*. We found evidence of consolidation of the lower two-thirds of the left lung, viz., dulness on percussion and marked tubular breathing. The heart sounds were not quite clear, but the pulse was so frequent that I could not decide whether there was, or was not, an endocardial murmur. He was ordered to continue the sixteen ounces of wine. In the evening the house-surgeon thought the patient still weaker,—his pulse was 132,—and ordered him to have an ounce of brandy every hour during the night, instead of the wine. He rallied under this treatment, and the next day was somewhat better. The pulse was 120; he was nearly free from pain, but there was a miliary eruption all over his arms, chest, and back. He had continued the brandy during the night, and resumed the wine in the morning. He was taking a good deal of nourishment in the shape of milk and beef-tea. When I saw him in the middle of the day, I thought he would probably improve more rapidly with a stronger stimulant than the wine, and I ordered him half an ounce of brandy every hour. I reduced the opium to a grain every eight hours, and I gave him two eggs in addition to his beef-tea and milk.

On the 19th, the pulse was 112; the respirations were 40. He had expectorated some adhesive rust-coloured sputa. There was no change in the physical signs. On the 20th, the pulse was 112, the respirations were 28; and on the 22nd they were 108 and 34. The respiration at the left base was less tubular, and coarse crepitation was heard. On the 25th he had passed a very good night; pulse 104, soft and regular; respirations 32. He perspired very little; the expectoration was scant, but very adhesive. On the 24th, the opium was diminished to a grain and a half every night. On the 26th, the brandy was reduced to ten ounces daily, and a chop was ordered. On the 28th the stimulants were further reduced to eight ounces. He was rapidly improving, and the physical signs were becoming normal. The hands were free from swelling, and almost entirely free from pain. On the 29th, I ordered him a grain of quinine three times a day, and ten ounces of port wine instead of the brandy. On the 1st of July, the pulse had fallen to 84. I need not follow out the case in detail. Steady improvement took place, under the influence of stimulants, which were gradually diminished, and of tonics, —quinine, iron, and cod-liver oil. He was discharged from the hospital on August 5th.

As the patient became convalescent, and the circulation became more quiet, we found that there was a distinct endocardial systolic murmur; but the lung sounds became quite healthy, so that, although there had been previous symptoms of tubercular disease, and hæmoptysis had occurred on two occasions, we hoped that, at all events if phthisis existed, it was but little advanced; for had it been otherwise, we should probably not have had so favourable an issue to our treatment.

Now let me call your attention to some of the features of the case. It was an instance of severe rheumatic fever, complicated with acute pneumonia, involving a large portion of one lung. The patient was treated simply by stimulants, opium, and free nutrition, and he made a satisfactory recovery. The quantity of stimulants given was somewhat large, twelve ounces of brandy a day for several days, and yet you saw that no effects of an intoxicating character were produced, there was no disturbance of the mental faculties, nor were the functions of the digestive organs interfered with. The inflammation of the lung was, not only, not increased under their use, but it gradually and steadily subsided; the profuse perspiration ceased, the inflammation and pain in the joints disappeared, and the endocarditis had a favourable issue.

This case is, I think, a very instructive one, and illustrates very powerfully the important principle I have endeavoured to impress on your minds, viz., that, in the use of therapeutic measures, you must have regard, not so much to the individual disease, of which your patient is the subject, as to the general symptoms,—the pathological conditions, in fact,—which that disease has produced. The prominent feature in the case was the rapid and feeble pulse; and although the patient was suffering from acute rheumatism, I did not hesitate to administer stimulants, and the result proved that they were by no means inimical to the rheumatic inflammation; for, looking at the case simply as one of rheumatic fever, we find that convalescence was fully established in a month from the commencement of the attack—not a very unsatisfactory result, taking the severity of the symptoms into consideration.

But you must not draw, from the result of this case, the inference that rheumatic fever should always be treated with large doses of stimulants, although it is quite certain that some cases of this affection are much benefited by the exhibition of them. You probably recollect the case of John M., who was in the wards in April and May last. This man had acute rheumatic inflammation of the joints, with a very quick and feeble pulse. I treated him for several days with alkalies and bark; but the inflammation did not diminish, and the debility increased.

I then omitted all medicines, and gave twelve ounces of port wine daily, with an opiate at night. The patient began at once to improve, and made a somewhat rapid recovery. He was admitted on the 29th April, and on May 6th we commenced the treatment by stimulants alone. The pulse was then 120, and very weak; the tongue was dry and brown; there was a good deal of swelling about the joints, and the pain was very severe. On the 10th of May the pulse had dropped to 90; on the 13th it was 80, and the pain and swelling of the joints had disappeared —in fact, the man was convalescent. No relapse followed.

Let me read to you the notes of another case of pneumonia, occurring during an acute rheumatic attack. This was a mild case. The patient was never in any danger, although a considerable portion of one of his lungs was implicated. The treatment was in nowise different from that which I should have adopted for the joint affection, and no stimulants were given throughout the attack. There were, in my opinion, no symptoms calling for their exhibition.

George A., aged 32, was admitted into the hospital on the 9th of January, 1865, his illness having commenced two days previously. The man was a shipwright, and had had rheumatic fever twenty years before he came to us. We found all the larger joints more or less swollen, red, painful, and tender to the touch. The patient was restless, and covered with perspiration. The pulse was 92. The action of the heart was regular, but the impulse was forcible, and the area of cardiac dulness was increased. There was, moreover, a systolic bruit heard at the base.

From the history of the patient, from the character of the bruit, and especially from the evidence of hypertrophy of the heart, I concluded that the murmur was the result of old standing valvular disease, and not of recent endocarditis. I put the patient on twenty grains of bicarbonate of potash every three hours, and a grain of opium three times a day, with beef-tea and milk for diet. We examined the urine, and found it free from albumen. On the 11th of January, the rheumatic symptoms had improved, but the man complained of dyspnœa, and shooting pains about the chest. The pulse was only 80. On the 14th, I made a careful examination of his chest. I found the cardiac murmur as before; there was dulness at the lower part of the left lung, with bronchial breathing and broncophony.

Here then, we had an attack of pneumonia. I did not think it necessary to change the treatment further than to order a blister to the chest.

On the 16th, the urine was found alkaline, and the quantity of potash was diminished. The patient continued to improve, but on the 19th his pulse rose to 100, and I ordered him some bark with the potash. On the 23rd he was able to raise himself in bed. There was still dulness and tubular breathing at the back of the left lung. On the 27th he was ordered chop diet and quinine. He steadily improved, and on the 4th of February the percussion and respiration sounds were normal. He was discharged, well, on the 10th of February, the murmur remaining about the same as it was on his admission.

I must give you one caution before I conclude this lecture, a caution which both these cases serve to show the importance of, viz., not to omit to make a careful examination of the chest, from time to time, in your rheumatic patients, even if there are no active symptoms of lung or heart complication. In both my cases the pneumonia must have existed for a few days before I discovered it, and nothing but a physical examination would have enabled me to ascertain its existence. So insidious is the disease, sometimes, that unless you are on the watch you may altogether overlook it.

CHAPTER VIII.

PNEUMONIA — TREATMENT — ABSTRACT OF CASES — PNEUMONIA OF CHILDREN — PNEUMONIA OF THE AGED — TABLE.

(CLINICAL LECTURE.)

GENTLEMEN,—I wish to call your attention to-day to the Table, which contains a short abstract of forty-four consecutive cases of acute pneumonia which have been treated in this hospital under my care.

The Table presents the leading features of each case. The first column gives the number of the case; the second, the initials of the patient; the third, the age, sex, and occupation; the fourth, the previous health, as far as could be ascertained; the fifth, the date of the commencement of the attack, generally marked by the occurrence of rigors; the sixth, the date of admission into the hospital; the seventh, the side, and extent of lung, involved; the eighth, the frequency of the pulse; the ninth, the frequency of the respiration; the tenth, the treatment; the eleventh, the date of convalescence, viz., the period when all active symptoms had subsided, when the pulse had fallen to a natural, or nearly natural standard, 60 to 80, and when the patient was able to take solid food. This column also gives the number of days the patient had been under treatment when convalescence was established, and the number of days which had elapsed from the commencement of the attack. The twelfth column gives the date of discharge, and the number of days the patient was in the hospital; whilst the thirteenth gives the result, with observations on the complications or peculiarities of the case.

First, as to the age of the patients. Under 10 years of age there was one case; between 10 and 20 years, there were six cases; between 20 and 30 years, there were twenty-three cases; between 30 and 40 years, there were eight cases; between 40 and 50 years, there were five cases; and between 50 and 60 years, there was one case. All the patients were males, with the exception of two. Many of the patients were strong, robust-looking men, whose previous health had been good, and in whom the disease had existed for a few days only before admission, the attack being distinctly traceable to exposure to wet or cold, or to both.

The list includes a large proportion of sailors, but also men engaged in town work, porters, hawkers, &c.

Of the forty-four cases, the pneumonia was single in thirty-five cases, involving from one-half to the whole of the lung; it was double in nine cases.

Of the single cases, the right lung was the seat of the disease in eighteen cases, the left in seventeen cases.

Of the double cases, the left lung was most involved in six cases, the right in one case. Both lungs were equally involved—viz., one-half, in two cases.

With reference to the treatment adopted. In no instance was venesection practised. Only three patients were cupped, viz., No. 2, to 12 oz., No. 9, to 8 oz., and No. 7, to 8 oz. In only three cases were leeches applied, viz., in No. 3, twelve; in No. 5, eight; and in No. 7, twelve. In Nos. 1 and 13, leeches had been applied before admission.

Whenever antimony was used, it was in small doses, varying from one-twelfth to one-fourth of a grain, except in Nos. 6 and 2, in which it was given in doses of three-fourths of a grain, and a grain, respectively.

In the majority of cases no antimony was given.

In a large proportion of the cases some alcoholic stimulant was given early in the disease. In twenty-four cases it formed the main therapeutic agent, and in some of the most severe of these no other medicine was given. In six of the other cases it was given after a few days' treatment by other means, antimony, etc.

The stimulants were given at regular intervals—every hour, or every two, three, or four hours, frequently with food, beef-tea, or milk. In the most severe cases, those marked with a very rapid pulse and great dyspnœa, brandy was given every hour, or every hour and a half.

Mercury—calomel with opium—was not given in any case. In No. 10 I gave blue pill twice a day for six days; but no soreness of the gums was produced. In no other instance was mercury given, except as a purgative, in combination with some other drug, at the commencement, or during the course of the treatment.

In every case nutrients were allowed freely, viz., beef-tea and milk from the commencement of treatment, and solid food as soon as the appetite enabled it to be taken.

Of the other substances used but little need be said. Carbonate of ammonia and chloric ether were given in most of the cases treated by the early administration of alcohol, and in some cases ipecacuan wine was used.

Now let me draw your attention to the results. Of the forty-four cases, one died. In this case, No. 7, the patient seemed to have rallied under the influence of stimulants, his pulse had fallen to 80, and his respirations to 20. The acute symptoms of the pneumonia had subsided, and convalescence appeared to be established. Effusion into the pleura, however, took place somewhat suddenly, to a large extent, and death soon followed.

The following statement shows the duration of the disease from the commencement of treatment to the time of convalescence in the cases that recovered.

In one case convalescence was established at the end of the third day of treatment; in six cases, at the end of the fourth day; in seven cases, at the end of the fifth day; in four cases, at the end of the sixth day; in five cases, at the end of the seventh day; in seven cases, at the end of the eighth day; in three cases, at the end of the ninth day; in one case, at the end of the tenth day; in three cases, at the end of the eleventh day; in one case, at the end of the fourteenth day; in three cases, at the end of the sixteenth day; and in one case, at the end of the twenty-fourth day.

The average duration of these cases, from the commencement of treatment to the period of convalescence, was 8⅓ days.

But it is important to ascertain how soon convalescence was established from the commencement of the attack, as well as from the commencement of treatment. Most of the patients had been ill for a few days before they were admitted into the hospital.

In thirty-four cases I was able to ascertain the date of the commencement of the attack. Of these, three cases were convalescent at the end of the sixth day of the attack; two cases, at the end of the seventh day; three cases, at the end of the eighth day; four cases, at the end of the ninth day; three cases, at the end of the tenth day; six cases, at the end of the eleventh day; two cases, at the end of the twelfth day; two cases, at the end of the thirteenth day; two cases, at the end of the fourteenth day; one case, at the end of the fifteenth day; two cases, at the end of the sixteenth day; one case, at the end of the seventeenth day; one case, at the end of the nineteenth day; one case, at the end of the twenty-fifth day; and one case, at the end of the twenty-sixth day. This gives an average of nearly 11 days for the thirty-four cases. In the remaining ten cases the date of attack could not be ascertained.

In taking the average of the number of days during which the patients were in the hospital, it must be borne in mind that, for the most part, they were kept in, not only till they had fully recovered from their attack, but till they had gained sufficient strength to be able to resume their work. Again, a few of the patients remained in the hospital several weeks, in consequence of their health being impaired from other causes besides the pneumonia. Thus, No. 41, who was 53 days in the house, had a severe attack of rheumatic fever. No. 34, who was 69 days in the house, was kept in, in consequence of tubercular symptoms. No. 30, who was in 52 days, suffered from gangrene of the lung, and, consequently, had a protracted convalescence. No. 24 was 49 days in the house. Although he soon recovered from the pneumonic symptoms, he regained his strength very slowly. No. 17 was in the house 43 days. This patient had attempted to drown herself; her health was seriously impaired, and she convalesced slowly. In No. 14 there was debility with emphysema of both lungs; this patient remained in the house 44 days. Excluding these six cases of very protracted residence in the hospital from the causes mentioned, we have, as the average of the remaining thirty-seven cases, 19⅔ days, and including the six cases, we have a general average of 24 days.

These are all the cases of acute pneumonia uncomplicated with advanced organic disease, as phthisis, Bright's disease, or disease of the heart, which have occurred in my practice in this hospital. They were all marked by the characteristic physical signs as well as the general symptoms of pneumonia, and the progress of each case was carefully noted from day to day.

The results of these cases, especially when taken in conjunction with others, of which statistics have been given, tend to prove that pneumonia is far from being the fatal malady it was formerly supposed to be, and that, under a treatment which consists in supporting the patient, and in abstaining from depletory, or depressing measures, its mortality is low.

I have purposely abstained from including in the table any but hospital cases, as these alone are available for public reference. It may, perhaps, be thought by some, that the kind of treatment adopted in these cases is not as well suited for patients met with in private practice, as for those who have to seek admission into a public institution; that the latter, from a variety of causes, come to us with a constitution more or less impaired, and thus, that they necessarily require a tonic and stimulating treatment, and will not bear the so-called antiphlogistic remedies; whilst the former, whose previous health has been less impaired, require a somewhat opposite treatment. My own experience does not accord with this view. In the cases of pneumonia which I have treated in private practice, either alone, or in conjunction with other medical practitioners, I have pursued a line of practice similar to that which I have adopted in the cases referred to in the table. In fact, the attacks of pneumonia which we meet with amongst the better classes of society, brought on, as they often are, by circumstances which have been for some time undermining the general health, require a stimulating and restorative treatment quite as much as those of our hospital patients. I have detailed in a previous chapter some instances in which stimulants proved injurious, whilst antimony exerted a beneficial effect. To distinguish between these cases sometimes becomes a difficult problem. When the pulse is quick and feeble, there should be no hesitation in the use of stimulants, but in some cases the pulse, although quick, possesses a sharpness, which indicates the propriety of withholding stimulants altogether.

But in considering the value of the alcoholic or other remedies which we give our patients, we must never forget the influence of rest in bed and proper nursing. In a large proportion of cases of disease, the rest in bed and the proper nursing, including the regulation of the atmosphere and temperature of the room, and the administration of nourishment, constitute a large part of our treatment, and we are too apt, in speaking of the effects of different remedies in a particular disease, to forget this important element of cure. The change which has taken place in the practice of medicine of late years consists quite as much in the substitution of a more liberal allowance of food, instead of the absolute starvation of former years, as in the almost total abandonment of blood-letting, and the use of stimulants in lieu of the frequent venesections and the large doses of depressing remedies then prescribed. The results of recent observations as to the effects of various remedies used in disease, may shake our confidence in the specific virtues of these substances, but they by no means alter our opinion of the value of treatment.

In watching your cases of pneumonia, there are two symptoms to which you must pay special attention, viz., the pulse and the respiration. As these diminish or increase in frequency, so may you argue favourably or unfavourably of your patients. If you find, when you are giving stimulants, that the pulse falls from day to day, you have an indication that you are pursuing a proper mode of treatment; but if, on the other hand, the pulse rises, you must seriously consider whether they ought not to be withdrawn. This rise, however, may be an indication that you are not giving enough stimulants, and by increasing the quantity you may bring down the pulse.

But before I conclude, I must draw your attention to some other points in connection with pneumonia. We do not often have an opportunity in the hospital of seeing a case of the disease in a child, and I wish, for this reason, to refer you to the only case reported in the table, under the age of ten years. In private practice you will often meet with cases of this kind, but, for obvious reasons, you will see but few in our wards.

In very early life pneumonia is a very fatal malady, but in children between the ages of five and fifteen years it is much less so. Of 212 cases reported by M. Barthez, occurring in the Hôpital St. Eugénie, Paris, between the ages of two and fifteen years, only two died.

The point, however, to which I wish specially to draw your attention is the treatment which you should adopt. It used to be the practice, and, I believe, it is still with some physicians, to give antimony and calomel, and to apply leeches to the chest; but it is a practice I have never adopted. In almost all cases of inflammation within the chest in children, I have resorted to a more or less stimulating treatment, and I have found it attended with the most satisfactory results. The fact is that children soon get through the disease if they are not submitted to depressing measures; further, they bear stimulants very well, and under their use convalescence is rapidly established.

Let me read to you the case of William T., which some of you will perhaps recollect. The boy was six years of age, and was admitted into K ward, under my care, on the 13th of May of the present year (1867). He had had a cough for some days, but nothing had been done for it in the way of treatment. I first saw him on the 14th of May. He had a quick pulse, a hot skin, and a good deal of dyspnœa. On examining his chest, we found distinct crepitation at the back of the right lung, with dulness, bronchial breathing and bronchophony over the lower half; in fact, we had evidence of confirmed pneumonia,—of hepätization of a considerable portion of one lung,—the other lung being healthy. I ordered him ten minims of aromatic spirits of ammonia, and the same quantity of chloric ether, in cinnamon water, every four hours, and I gave him, in addition, a teaspoonful of brandy every three hours. On the following day the pulse was 120, and there was crepitation over nearly the whole of the back of the left lung. I continued the brandy and the mixture, with the addition of five minims of ipecacuan wine to each dose. On the 16th, the pulse was 76, the crepitation was of a coarse character, and there was less dulness. On the 17th, the pulse was 72: and on the 18th, 80. The child was much better; in fact, getting well rapidly. The treatment was persevered with, more food being allowed as convalescence was established. On the 21st of May—viz., at the end of the seventh day of treatment—the breath and percussion-sounds were normal over the left lung.

Now this case was not a very severe one, although it involved a large portion of one lung. It was an average case of pneumonia, such as you will often meet with in children. I think it very probable that the child would have made a good recovery if neither medicines nor stimulants had been given, if we had trusted simply to rest in bed and the exhibition of nutrients. But I also believe that the case would have been more protracted, and that convalescence would have come much later.

There is no instance in the table of pneumonia in the aged. The age of the oldest patient was 55 years. But pneumonia is by no means infrequent in old age. It often comes on very insidiously, and is only to be detected by a careful physical examination. The mortality in old people has been very great, especially when blood-letting has been practised. Let me, therefore, caution you, if it is necessary after what I have said, against the use of the lancet, or even of the cupping-glasses, or of leeches, in the treatment of these pneumonic attacks in the aged. I have nothing special to add to what I have already said on the general principles of treatment in reference to such cases as I am alluding to now. You may give stimulants freely in some instances with the most marked benefit, and you must take care that as much nourishment is given as possible. Under a treatment of this kind you will have, I venture to say, far more successful results than if you were to pursue depletory measures in individuals already enfeebled by age.

There is another case in the table to which I wish to call your attention, as it has been quite recently under observation, and you have all had an opportunity of seeing it. Moreover, it offers some features of peculiar interest. It was a case of acute pleuro-pneumonia, resulting directly from the patient being immersed in the water. The man's name was Thomas F., he was 44 years of age, and previously in good health. He was one of the firemen of the Greek vessel on board of which an explosion took place a few weeks ago. He was down in the stoke-hole at the time the accident happened, and as the water rushed in at the bottom of the vessel he was carried up by it. He had to swim to save his life, and he was struck violently on the right shoulder and back by a piece of wood, or a portion of the machinery. When brought to the hospital on the 29th November, he was placed in the surgical wards, but as no fracture of the ribs could be detected, and as symptoms of pneumonia came on, he was transferred to my care on the 1st December, but I did not see him till the 2nd.

The following are the notes of the case taken at noon on Dec. 1st. Pulse 110.—Great dyspnœa, with severe lancinating pain on the right side of the chest, increased on inspiration; skin hot; face flushed; deficient movement of right side. He was ordered a saline with squills and ipecacuan wine. At night the pulse had risen to 120, and the respirations were 60. The pain in the side was very acute, and distinct crepitation was heard over the right lung. Mustard and linseed-meal poultices were ordered to the side. On the 2nd December, at one p.m., I found the pulse 84, and the respirations 48. There was still acute pain on the right side, with dulness and crepitation over the lower half of the right lung—most marked in the infra-axillary region—sub-crepitant *râles* were heard at the back of the left lung. The pain in the side was very great, and quite of a pleuritic character, but I could make out no friction-sound. I ordered a grain of opium to be given three times a day, and five grains of carbonate of ammonia, with fifteen minims of ipecacuan wine, every three hours. I also ordered beef tea and milk for diet. On the 3rd December, the patient was going on favourably, and the pain and dyspnœa had diminished. I made no alteration in the treatment, except to order a hot linseed-meal poultice to be constantly applied to the right side. On the 4th, the pulse was 84, the respirations were 42. There was less pain. There was dulness and absence of breath-sounds at the base of the right lung. I ordered a tablespoonful of brandy every three hours, the ammonia mixture to be given every four hours only, and a grain and a half of opium at night. On the 5th, the pulse was 80, the respirations were 30. There was well-marked friction-sound below the right nipple, with dulness over the lower half of the lung, and absence of breath-sounds at the base. On the 7th, the patient was free from pain, even on deep inspiration; but his cough was troublesome. The pulse was 80, and there was no dyspnœa. On examining his chest we found much less dulness on percussion; the breath-sounds were audible at the base, with crepitation, and there was bronchial breathing, with pleuritic friction-sound higher up. The opium was omitted. The brandy was continued, and meat diet was ordered, with some carbonate of ammonia, and sulphuric ether. He improved rapidly from this date, and left the hospital well, on the 12th December.

The case is interesting as an instance of pleuro-pneumonia involving a considerable portion of one lung, occurring in a previously healthy man, from the direct effects of wet and cold. You saw that from the commencement a moderately stimulating treatment was adopted, and nutrients were freely allowed. Again, you saw how acutely the man suffered from pleuritic pain, and how it yielded to the exhibition of opium, and the application of hot poultices, without any local abstraction of blood.

TABLE OF CASES.

TABLE OF CASES OF ACUTE PNEUMONIA, WITH ABSTRACT OF TREATMENT.

No.	NAME.	Sex, Age, and Occupation.	PREVIOUS HEALTH.	Commencement of Attack.	Date of Admission.	Amount of Lung involved.	Pulse.	Respiration.	TREATMENT.	Date of Convalescence.	Date of Discharge. Number of days in hospital.	RESULT. REMARKS.
1	L. J.	M., 47, Sailor.	Good, a stout, muscular man.	Ill about ten days before admission.	Mar. 14, 1860.	⅔ Left.	92	Dyspnœa.	Leeches before admission. ⅛ gr. antimony with saline every three hours, from March 14th to 21st. Blister on 16th.	March 18, end of 4th day of treatment and 14th of attack.	March 22. 8 days.	Recovered.
2	W. E.	M., 29, Sailor.	Good. A stout, muscular man.	Mar. 23, 1860.	Mar. 29 "	¾ Left. Base right.	110	40 Much dyspnœa.	One gr. antimony with saline every three hours, from March 29th to April 2nd, then three times a day until April 3rd. Cupping to 12 oz. on 29th March.	April 3, end of 5th day of treatment and 11th of attack.	April 7. 9 days.	Recovered.
3	E. W.	M., 19, Sailor.	Good.	April 21 "	Apr. 23 "	¾ Right. Pleurisy.	106	40 Dyspnœa.	April 24th, 12 leeches. ⅛ gr. antimony with saline every four hours, from April 25th to 28th; 26th, 8 leeches, then saline alone; 4 oz. wine from May 2nd to 5th, then 6 oz. Blister on May 2nd, and again on May 17th. Iodine to chest, and subsequently iod. pot. internally.	May 17, end of 24th day of treatment and 26th of attack.	May 25. 32 days.	Recovered. This case was complicated with pleurisy, which delayed convalescence.
4	C. H.	M., 32, Fireman.	Good.	June 1 "	June 4 "	⅔ Left.	88	Dyspnœa.	Saline every 4 hours for one day, then saline with ⅛ gr. antimony every four hours till June 8th, every eight hours till June 9th, and 4 oz. wine daily; 8 leeches on the 5th; blister on the 8th.	June 9, end of 5th day of treatment and 8th of attack.	June 26. 22 days.	Recovered.
5	J. W.	M., 26, Labourer.	Good.	June 5 "	June 8 "	½ Left.	92	36	¾ gr. antimony with saline every four hours, from June 8th to 11th. Blister.	June 11, end of 3rd day of treatment and 6th of attack.	June 19. 11 days.	Recovered.

									proved under the stimulants. On 13th, his pulse was 80; respiration, 20. He became worse after this, and died on 25th.			
8	G. J.	M., 26, Sailor.	Not good.	Seventeen days before admission.	Feb. 13 ,,	⅓ Right. Pleuritic effusion.		Dyspnœa.	12 leeches. Blister. Saline.	Feb. 21, end of 8th day of treatment and 25th of attack.	Feb. 26. 13 days.	Recovered.
9	H. H.	M., 17, Sailor.	Good.	Apr. 21, 1861.	Apr. 24 ,,	Upper lobe. Right.	116		⅙ gr. antimony every three hours, from April 25th to 27th; every four hours to 28th; every six hours to May 1st; then omitted. One pint porter daily.	April 28, end of 4th day of treatment and 7th of attack.	May 9. 15 days.	Recovered.
10	W. K.	M., 24, Labourer.	Ailing for some weeks		May 6 ,,	½ Left.	100	24	Blister. 6 oz. wine daily. 2 grs. blue pill twice a day.	May 10, end of 4th day of treatment.	May 13. 7 days.	Recovered.
11	T. D.	M., 28, Porter.	Good. A strong, muscular man.	July 10 ,,	July 12 ,,	¾ Left.	120	40	Cupped to 8 oz. on 13th. ¼ gr. antimony every three hours, from 13th to 14th; then every four hours to 16th; then three times a day to 18th; then stopped. Blister on 17th. Quinine on 27th.	July 23, end of 11th day of treatment and 13th of attack.	August 3. 22 days.	Recovered.
12	L. S.	M., 24, Sailor.		Eight days before admission.	Dec. 17 ,,	Right. Upper lobe.	96	40	Small doses antimony for three days. 6 oz. wine.	Dec. 28, end of 11th day of treatment and 19th of attack.	Jan. 2, 1862. 15 days.	Recovered. Pneumonia of apex (tubercular?)
13	T. R.	M., 21, Hawker.	Intemperate.	Dec. 25, 1861.	Dec. 28 ,,	¾ Left. Small part Right.	116	60	Six leeches before admission. ¼ gr. antimony every two hours for one day; every four hours for two days; every six hours for one day; laudanum for delirium; brandy and wine. One blister.	Jan. 5, end of 8th day of treatment and 11th of attack.	Jan. 21, 24 days.	Recovered. Complicated with delirium. A man of intemperate habits.

No.	Name.	Sex, Age, and Occupation.	Previous Health.	Commencement of Attack.	Date of Admission.	Amount of Lung involved.	Pulse.	Respiration.	TREATMENT.	Date of Convalescence.	Date of Discharge. Number of days in hospital.	Result. Remarks.
14	M. McC.	F., 25.	Weak.	A week before admission.	Jan. 1, 1862.	½ Right. ½ Left. Bronchitis.	116	60	⅛ gr. antimony every four hours, from Jan. 2nd to 6th; improved. Antimony omitted for one day. Relapse. Repeated from 7th to 11th. Stimulants.	Jan. 11, end of 10th day of treatment and 17th of attack.	Well Jan. 23. Kept in till Feb. 13 on account of weakness 44 days.	Recovered. A thin, spare woman. Case complicated by bronchitis and emphysema.
15	C. W.	M., 26, Sailor.	Good. A strong, muscular man.	Apr. 24, 1862.	Apr. 26 ,,	⅔ Left.	108		¼ gr. antimony every three hours on 26th; every four hours on 27th; omitted on 28th; blister on 29th.	May 1, end of 5th day of treatment and 7th of attack.	May 9. 13 days.	Recovered.
16	G. C.	M., 25, Sailor.	Good.	Four days before admission.	Jan. 29, 1863.	⅔ Left.	109		⅛ gr. antimony every three hours, from Jan. 30th to Feb. 1st; three times a day from 1st to 3rd; blister 31st Jan.	Feb. 3, end of 5th day of treatment and 9th of attack.	Feb. 12. 14 days.	Recovered.
17	E. H.	F., 40.	Not good.	Oct. 8, 1863.	Oct. 8 ,,	Right. Bronchitis.	100		Carb. ammonia and sulph. ether. 8 oz. wine daily, and 3 oz. brandy subsequently. Bark.	Oct. 24, end of 16th day of treatment and of attack.	Nov. 20. 43 days.	Recovered. Case of broncho-pneumonia, following immersion. The patient had been put into the hot bath, when recovered from the water and before she was taken to the hospital, she had attempted suicide.
18	L. A.	M., 29, Sailor.		Nov. 22 ,,	Nov. 22 ,,	⅔ Left. Base of	132	Great dyspnœa.	Admitted on 20th Nov. with bronchitis. Stimulants ordered. On 22nd, well-	Nov. 28, end of 6th day of	Dec. 11. 19 days.	Recovered.

									on 24th, and $\frac{1}{4}$ gr. antimony with saline given every four hours. Improvement. Antimony diminished on 26th, and stopped on 28th.			
19	W. S.	M., 30, Labourer.	Good.	Mar. 21, 1864.	Mar 23, 1864.	$\frac{3}{4}$ Right.	120	40	$\frac{1}{4}$ gr. antimony every four hours, from March 23rd to 25th; subsequently ammonia, ipec. and wine. Blister on 23rd.	March 27, end of 4th day of treatment and 6th of attack.	April 1. 9 days.	Recovered.
20	P. F.	M., 36, Carter.	Intemperate.	Aug. 8 ,,	Aug. 8 ,,	$\frac{1}{2}$ Left.	120	32	March 9th, a grain of opium three times a day, $\frac{1}{10}$ gr. antimony every four hours; 10th, opium stopped; ipec., laudanum and antimony to 13th; then wine (6 oz.) and carb. ammonia; 17th, quinine.	Aug. 16, end of 8th day of treatment and 8th of attack.	Sept. 7. 30 days.	Recovered. Gained strength slowly. Of intemperate habits.
21	G. A.	M., 32, Shipwright		Jan. 11, 1865.	Jan. 11, 1865.	$\frac{1}{3}$ Left. Acute rheumatism.	92		Alkalies (pot. bicarb.). Opium at night. 19th, bark with potash; 27th, quinine.	Jan. 27, end of 16th day of treatment and 16th of attack.	Feb. 10. 30 days.	Recovered. Admitted with acute rheumatism on Jan. 8; pneumonia on Jan. 11.
22	D. M.	M., 55, Fireman.	Good.	Jan. 21 ,,	Jan. 23 ,,	$\frac{1}{2}$ Left. $\frac{1}{4}$ Right.	120	40	23rd, noon. Stimulants, brandy and ammonia; became worse at night. $\frac{1}{18}$ gr. antimony with saline and ipec. every three hours to night of 24th; then every four hours; decided improvement. Mixture stopped on 25th, and an oz. of port wine given every three hours; became worse. On 27th, antimony resumed every two hours; 28th, every four hours; 30th, three times a day; improved; 31st, antimony omitted; 6 oz. wine.	Feb. 1, end of 9th day of treatment and 11th of attack.	Feb. 22. 30 days.	Recovered. A somewhat spare Frenchman. Stimulants aggravated the symptoms. Antimony gave relief.
23	J. D.	M., 18, Sailor.	Good.	Feb. 19 ,,	Feb. 24 ,,	$\frac{3}{4}$ Left.	120	76	Half an ounce of brandy every two hours, from Feb. 24th to March 4th; then 5 oz. daily to 6th; then 6 oz. port wine, carb. ammonia and ipec. from Feb. 24th to March 7th. Quinine.	March 4, end of 8th day of treatment and 13th of attack.	March 17. 21 days.	Recovered.

No.	NAME.	Sex, Age, and Occupation.	PREVIOUS HEALTH.	Commencement of Attack.	Date of Admission.	Amount of Lung involved.	Pulse.	Respiration.	TREATMENT.	Date of Convalescence.	Date of Discharge. Number of days in hospital.	RESULT. REMARKS.
24	J. M'N.	M., 20, Labourer.	Good.	Nov. 13, 1865.	Nov. 17, 1865.	$\frac{1}{2}$ Right.	116	44	An ounce of port wine every three hours, from Nov. 18th to 25th; then 6 oz. daily; ipec. and morphia to 25th, then quinine; 20th, blister.	Nov. 25, end of 8th day of treatment and 12th of attack.	Jan. 5, 1866. 49 days.	Recovered. Gained strength slowly.
25	C. A.	M., 40, Sailor.		Nov. 14 ,,	Nov. 18 ,,	$\frac{3}{4}$ Right.	120	48	For one day $\frac{1}{12}$ gr. antimony every three hours; omitted and stimulants given, viz., 19th, 6 oz. port wine; 20th, 8 oz.; 21st, 12 oz.; 29th, 10 oz.; Dec. 9th, 6 oz.; ipec. and morphia from 19th; quinine Dec. 2nd. Two blisters.	Nov. 25, end of 7th day of treatment and 11th of attack.	Dec. 16. 28 days.	Recovered.
26	B. M.	M., 27, Labourer.	Good.	Jan. 5, 1866.	Jan. 9, 1866.	Upper part Right.	120		Carbonate of ammonia, ipec. and chloric ether, and 6 oz. port wine, daily, to Jan. 22nd, then quinine. One blister.	Jan. 14, end of 5th day of treatment and 9th of attack.	Jan. 30. 21 days.	Recovered.
27	T. C.	M., 23, Labourer.		Ailing for some time.	Jan. 25 ,,	Right. Pleuro-pneumonia effusion.	108		Carbonate of ammonia, chloric ether, and squills. Two blisters. Feb. 3, quinine and wine.	Feb. 3, end of 9th day of treatment.	Feb. 28. 34 days.	Recovered.
28	M. J.	M., 28, Shoemaker		Three or four days before admission.	Feb. 1 ,,	$\frac{1}{2}$ Right. $\frac{1}{2}$ Left. Pleuritic effusion.	140	Great dyspnœa.	A tablespoonful of brandy every two hours, from Feb. 1st to 6th, then 4 oz. daily; carb. ammonia and squills, from Feb. 1st to 8th, then quinine. Blister on 2nd Feb.	Feb. 8, end of 7th day of treatment and 11th of attack.	March 3. 30 days.	Recovered. Very severe case of double pneumonia. Improved rapidly under stimulants.
29	A. M.	M., 30,			Feb. 2 ,,	$\frac{1}{3}$ Left.	112		A tablespoonful of brandy every two from Feb. 2nd to 15th; then	Feb. 10, end of 8th day of	Feb. 20 18 days.	Recovered.

												and very fœtid expectoration. Great prostration of strength
31	P. H.	M., 31, Groom.		Feb. 26, 1866.	Mar. 3 ,,	½ Left. Base right. Pleurisy.	116		Carbonate of ammonia, ipec. and chloric ether, from March 3rd to 10th; then quinine; 6 oz. port wine from March 4th to 12th.	March 9, end of 6th day of treatment and 11th of attack.	March 12. 9 days.	Recovered. Had systolic murmur at base of heart.
32	T. F.	M., 24, Sailor.	Good.	Eight days before admission.	Mar. 24 ,,	½ Right.	86		Carbonate ammonia and chloric ether. One blister.	March 31, end of 7th day of treatment and 15th of attack.	April 1. 8 days.	Recovered.
33	H. A.	M., 19, Carter.		A week before admission.	May 3 ,,	½ Right.	120		Tincture of iron, chloric ether, and quinine; 6 oz. wine daily. One blister.	May 8th, end of 5th day of treatment and 12th of attack.	June 9. 37 days.	Recovered.
34	R. H.	M., 26, Ship Carpenter.		May 25, 1866.	May 26 ,,	Whole of right lung. Upper part most affected.	140	50	27th May, ammonia and chloric ether; 29th, ammonia, saline, and ipec.; an oz. of wine every three hours; 30th, an oz. of wine every two hours; 31st, an oz. of brandy every three hours; June 1st, 8 oz. wine.	June 4, end of 9th day of treatment and 10th of attack.	August 2. 69 days.	Recovered. Suffered from cough for a month before admission. A case of pneumonia in a tuberculous (?) patient. Had hæmoptysis after acute symptoms had passed off, and was kept in hospital on account of his weak state and pthisical symptoms. Had delirium.

	Name.	Sex, Age, and Occupation.	Previous Health.	Commencement of Attack.	Date of Admission.	Amount of Lung involved.	Pulse.	Respiration.	TREATMENT.	Date of Convalescence.	Date of Discharge. Number of days in hospital.	Result. Remarks.
35	M. O'H.	M., 31, Labourer.		Ailing for some time.	Aug. 6 "	⅗ Left.	100		Carbonate of ammonia, ipec.; 8 oz. port wine daily; subsequently quinine.	Aug. 14, end of 8th day of treatment.	August 14. 8 days.	Recovered.
36	T. P.	M., 14, Sailor.	Good.		Oct. 2 1866.	Upper part. Left.	120		Ipec. and squills, from Oct. 2nd to 6th; no improvement; pulse kept up; on 6th, ammonia and 4 oz. port wine daily; improvement; 18th, quinine; 20th, 6 oz. wine; 23rd, iron and cod-liver oil.	Oct. 18, end of 16th day of treatment.	Nov. 12. 40 days.	Recovered. Was kept in the hospital on account of pthisical symptoms, but went out quite well.
37	R. R.	M., 23, Sailor.	Intemperate.	Oct. 15, 1866.	Oct. 18 "	½ Right.	120		Carbonate ammonia and chloric ether with saline every four hours, and 8 oz. brandy daily, from 19th to 27th; then 6 oz. brandy daily, to Nov. 1st; 25th, quinine.	Oct. 24, end of 6th day of treatment and 9th of attack.	Nov. 9. 21 days.	Recovered. Intermittent pulse; became strong and regular under stimulants.
38	R. R.	M., 43, Porter.	Good.	Jan. 28, 1867.	Feb. 1, 1867.	¾ Left.	110	38	Feb. 1st, 4 oz. brandy, ammonia, and chloric ether; 2nd, ½ oz. brandy every two hours; 3rd, ½ oz. brandy every one and a half hours; continued to 9th; then 6 oz. daily; subsequently, 12th, quinine and wine.	Feb. 8, end of 7th day of treatment and 10th of attack.	Feb. 26. 25 days.	Recovered. Complicated with delirium.
39	P. C.	M., 20, Militia Man.	Good.	April 25 "	Apr. 30 "	¾ Left. Base right.	110	52	⅙ gr. antimony with saline for two days; worse. May 2nd, a tablespoonful of port wine every two hours; carbonate ammonia and ipec.; improved.	May 6, end of 6th day of treatment and 11th of attack.	May 16. 16 days.	Recovered.
40	W. T.	M., 6.		Three or four days before admission.	May 13 "	⅗ Right.	120		A teaspoonful of brandy every three hours; ammonia and ipec.	May 17, end of 4th day of treatment and 8th of	May 21. 8 days.	Recovered.

									29th, wine, 10 oz., instead of brandy. During acute stage he took no medicine except opium. On 29th, quinine, and subsequently iron.			fever and endocarditis. Extreme exhaustion.
42	A. T.	M., 24, Sailor.			Aug. 28 „	¾ Right. Small part. Left.	96		29th August, 4 oz. brandy; 30th, 8 oz. wine; 31st, 12 oz. wine, carb. ammonia and bark.	Sept. 2, end of 5th day of treatment.	Sept. 17. 20 days.	Recovered.
43	J. M.	M., 28, Sailor.		Ill some time.	Sept. 18 „	½ Right.	114		Sept. 19, ammonia and bark; 26th, 6 oz. port wine; 28th, iodide of potassium, wine continued.	Oct. 1, end of 11th day of treatment.	Oct. 12. 24 days.	Recovered. Case of neglected pneumonia. Some dulness remained at time of discharge.
44	T. F.	M., 44, Fireman.	Good.	Nov. 29, 1867.	Dec. 1 „	⅔ Right. Pleurisy. Effusion.	120	60	Carbonate of ammonia and ipec., one gr. opium thrice a day; 4th, ½ oz. brandy every three hours, opium at night only; 7th, ammonia and ether.	Dec. 7, end of 7th day of treatment and 9th of attack.	Dec. 12. 13 days.	Recovered.

CHAPTER IX.

EMPHYSEMA OF THE LUNGS—ITS MORBID ANATOMY.

Opinions of earlier Pathologists. Views of Laennec. Frequency of the Disease. Importance of a knowledge of the Minute Structure of the Lungs. The different kinds of Emphysema. Vesicular Emphysema; its various Forms. Modes of Preparation of Lung-Tissue for Examination. Changes which take place in the Air-Sacs in the different Stages of the Disease. Condition of the Blood Vessels; of the Bronchial Tubes. Interlobular Emphysema.

PREVIOUSLY to the time of Laennec, pulmonary emphysema was, for the most part, considered by pathologists as produced by an infiltration of air into the areolar tissue of the lungs.* An imperfect knowledge of the anatomy of these organs led to the supposition, that a rupture of the air-cells would give rise to extravasation of air into the tissue by which they were connected with each other. An improved anatomical knowledge and further pathological inquiry have shown us that the quantity of areolar tissue in the lungs is very small; that extensive rupture of the air-cells may take place without infiltration of that tissue resulting; and that the only parts where such infiltration does occur are those, in which the above-named tissue surrounds the various lobules.

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* Descriptions of lungs which were large, and distended with air, and in some instances having dilated air-cells, will be found in the works of some of the earlier pathologists—Bonet, Morgagni, Floyer, Rusch, Valsalva, and Baillie; but no true definition of the disease was given before that of Laennec.

Laennec was the first to draw the distinction between the two kinds of pulmonary emphysema, viz., vesicular, and interlobular. The former he recognised as consisting in a dilatation or distension of the ultimate portions of the air-tubes. Subsequent researches have tended to prove the correctness of the French physician's observations in this respect; although the theory he advanced of the determining cause of the disease, as I shall have hereafter to show, has given way under an improved knowledge of the physiology of respiration, and more extended pathological inquiry.

Since the time when Laennec wrote, many able observers have directed their attention to this disease. Nevertheless, the information we possess with reference to it is insufficient to enable us to generalize with certainty on its essential nature; and there are numerous points in connection with its pathology and determining causes, on which investigations are necessary, in consequence of the diversity of opinion which exists thereon.

Of the great frequency of the affection in varying degrees of severity, no one who has been engaged in extensive public or private practice can entertain any doubt;* and, considering the gravity of the symptoms which the disease produces in its progress, and the tendency which exists for other organs to become secondarily involved, we cannot but admit the great importance of an early recognition of its existence, and of a more accurate knowledge of its essential nature.

In speaking of the disease, Lebert has the following remarks in his *Pathological Anatomy*:—"Few diseases have given rise in the last twenty years to so many theories, so many explanations, more or less conjectural; and as no one of them appears to us satisfactory, in the present state of science, it is the more essential to describe in a rigorously exact manner everything which relates to this affection, in order that for the future these materials may serve to establish doctrines more generally satisfactory on the subject."

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* In fifty-three cases of cholera examined by Louis, twenty-three of the individuals were found to be more or less affected with emphysema of the lungs, in various stages; nor was there any ground for the assumption that it had become developed during the fatal malady.

Again, writing on the same subject, Rokitansky observes:—"The conditions giving rise to the production of emphysema, and its pathogeny in general, although much labour has been devoted to the affection, are still far from clear." Further, Hasse, in speaking of the affection, and whilst admitting the influence of catarrh and hooping-cough as occasional causes of the disease, remarks:—The character and real import of emphysema must, however, be admitted to depend on other causes, as yet not thoroughly made out."

These quotations will be sufficient to indicate the uncertainty which at present exists with reference to our knowledge of the nature and pathology of this affection.

To the study of morbid anatomy and pathology, a knowledge of minute structure is essential; hence, the rapid strides which these sciences have made since the microscope has revealed to us the intimate arrangement of the various organs of the body. Important, however, as such knowledge is to the full appreciation of all diseased structure and function, its value becomes doubly manifest with reference to the disease now being considered; for there is probably no affection of the body, the symptoms and physical signs of which may be as easily explained as those of pulmonary emphysema, when once we are well acquainted with the structural changes and modifications by which the disease is accompanied.

For a description of the anatomy of the healthy lung, I must refer my readers to the first chapter of this work; and I now pass on to consider the changes which the lung-tissue undergoes in the progress of emphysema.

PULMONARY EMPHYSEMA is of two kinds:—

I. VESICULAR EMPHYSEMA.

II. INTERLOBULAR EMPHYSEMA.

The first is by far the more frequent and more important affection. I have rarely seen the second, except in advanced cases of the first kind.

I. *Pulmonary Vesicular Emphysema* exists in three forms, which, although they may differ as to their pathological causes, do not differ anatomically except as to the extent to which they involve the lung. We have, firstly, that which is the most partial, and confined to a few air-sacs, or to a single lobulette; secondly, that in which the whole of a lobule is affected; and thirdly, that in which the whole of one lobe of a lung, or, more frequently, the whole of the lung itself, is involved in the disease.

1. The first form of the disease, or *partial lobular emphysema*, is not often seen as an independent affection; but in lungs in which the second form exists, we occasionally meet with small patches of dilated air-sacs, especially along the margins of the lobes — patches which, clearly, only involve a few air-sacs, or, at most, a single lobulette. These portions resemble enlarged vesicles; and, as pointed out by M. Lombard, of Geneva, many years ago, have very much the appearance of the vesicles of pemphigus. They push outwards the pleura, so as to raise it above the level of the surrounding lung-tissue. They are not simple elevations of the pleura, as may be distinctly seen in injected preparations, when the blood-vessels belonging to the air-sacs may be observed ramifying beneath the pleura. Sometimes these partial emphysematous patches extend for some dis-

tance along the margin of the base of the lung, and they then have an appearance very like that of a row of beads.

2. The second form, or *lobular emphysema*, is that most frequently met with; in fact, in the majority of *post-mortem* examinations which I have seen, since my attention has been directed to this subject, I have found this form of emphysema more or less prevailing. It involves one or more lobules in different parts of the lung, and is especially found along the margin of the base, at the anterior border, and apex of the organ. This is the kind of emphysema so frequently met with in cases of phthisis; I have also seen it in cases of pneumonia where the lung has become consolidated; and in one instance of this latter kind the emphysema had all the appearances of having been recently produced.

In this form of emphysema it is easy to trace the different divisions of the lung; the boundary wall of the lobules has not given way, and there is no interlobular emphysema, except in some cases, and to a partial extent. Again, it by no means follows that all the air-sacs in an affected lobule are equally dilated; the most superficial ones, those especially beneath the pleura, suffer most. At the same time, it must be remarked that this unequal dilatation is sometimes more apparent than real, resulting from the mode of preparation to which the tissue has been subjected, viz., inflation and desiccation. During the process of drying, the air-sacs farthest from the surface collapse most; and thus they have the appearance of being smaller than those which are more superficial, without any real difference having originally existed between the two sets.*

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* Some anatomists have taught that the "vesicles" of the lungs are larger towards the surface than in the interior of the lungs. This statement seems to me altogether devoid of proof. It is quite true that, in a

The emphysematous lobules are seen on the surface of the lung, protruding beyond the level of the surrounding tissue; and along the margins of the lobes they often form projections of considerable size, in some instances becoming developed into the so-called "appendages."

3. The third form of pulmonary vesicular emphysema, or *lobar emphysema*, is by far the most important. As far as my observations go, it more frequently attacks both

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lung which has been inflated and dried, the "vesicles" which are situated at the surface, beneath the pleura, appear larger than those which are situated at a distance from it, and from the observation of this fact, in all probability, the statement has been made; but it must be borne in mind that this is the result of the collapse of the lung which takes place during desiccation. When a lung is inflated, and slowly dried, the surface is that which dries most quickly; and the *air-sacs* and *alveoli* there situated, being subjected to but little pressure, do not collapse to any very great extent, and thus they maintain a size approaching that they originally had; but with the interior of the lung the case is altogether different; the shrinking which necessarily takes place gives rise to considerable pressure on the air-sacs, and collapse of them results, producing an appearance of small "vesicles," from the process which is adopted, and not from any structural arrangement. From the examination of the human lung under various modes of preparation, and of the lungs of other animals, I have been led to the conclusion that there is no real difference of size between the superficial and deep air-sacs and alveoli of the lungs, nor yet between these structures in different parts of the organ; nor is it easy to see on what principle such a condition would be probable, or on what theory it could be expected.

These observations, with reference to the healthy lung, are equally applicable to the emphysematous organ; and the facts alluded to should be borne in mind, when we wish to make a comparison between the effects produced by the disease on the superficial and on the deep-seated air-sacs. It is, I think, highly probable that the circumstances I have stated have been overlooked by authors who have written on emphysema, and who have dwelt on the greater liability to distension of the superficial, as compared with the more deep-seated, lung-tissue.

lungs than one, and as well the lower as the upper lobes. It constitutes a most formidable affection, and often destroys life at an early period. The cases in which I have had an opportunity of seeing the disease after death, have been those of persons of adult age. Its features are very characteristic, and it is easily recognised during life. It consists of an emphysema of the whole pulmonary tissue of a lobe, or lung. The lung-substance has a peculiar doughy feel, pits on pressure, is wanting in healthy crepitation, and has a colour very closely resembling that of a calf's lung. The whole organ is increased in bulk. No collapse of the lung-tissue takes place when the walls of the chest are removed, in making a *post-mortem* examination; on the contrary, the lung often bulges forwards, as if its cavity were too small for it. It is also seen to overlap the heart, unless adhesions have occurred which prevent this taking place. The surface of the lung is not unfrequently marked by the impressions of the ribs. If a lung presenting this form of the disease be carefully removed from the chest, and inflated through its bronchus, it will be found that it may be distended to a very large size, without apparently any rupture taking place from the inflation. When the inflation is stopped, the air is expelled from the lung very slowly, and the collapse of the tissue is often very incomplete. The lung presents, when inflated, for the most part, a perfectly smooth and level surface, except in those cases where collapsed portions exist; or where portions of lung extremely emphysematous form projections or appendages. Frequently, the outlines of the lobules cannot be distinctly seen, in consequence of the rupture of their boundary walls having given rise to the production of interlobular emphysema. When this is the case, the air may be pressed laterally from one lobule to another.

In examining the emphysematous lung-tissue, to ascertain the anatomical changes of which it is the seat, I have adopted methods of preparation similar to those which I have made use of for the examination of the healthy lung, viz., first, inflation through a bronchial tube, and subsequent desiccation; and, secondly, injection of the blood-vessels with a coloured solution of gelatine, followed by inflation and desiccation. If we examine pieces of lung which have been prepared in either of these ways, under different degrees of development of emphysema, we are enabled to ascertain the exact condition of the pulmonary tissue in the progressive stages of the affection, and the extent to which it has departed from the normal type.

In the early stages of the disease we recognise a simple dilatation of the air-sacs, an increase in the size of the alveoli, and a diminution in the height of the alveolar walls, which, yielding with the distending cavities, become partially obliterated. As the disease progresses, the air-sacs become still more distended, and the alveolar walls, in some instances, completely obliterated, so as to give a regular and smooth appearance to the inner surface of the air-sacs, instead of the honey-combed appearance characteristic of their normal state. (See Plate V., figs. 11 and 12.) This distension of the air-sacs is necessarily attended with a divergence of the elastic fibres which enter into their composition, and with a general thinning of their walls—a condition which prepares the way for the next stage in the progress of the disease, viz., a perforation of the walls themselves. This, at first, is but slight; here and there a circular or oval opening may be seen in the membrane; as the disease progresses, these openings become more numerous, and larger; in some instances, the whole of the walls of the air-sacs and the septa of the alveoli being perfectly riddled with small openings, so that a horizontal section of the lung-substance has a general cribriform appearance. These openings are for the most part either circular or slightly oval. They exist in all parts of the walls, and are often seen in the septa between the alveoli, before the air-sacs are sufficiently distended to obliterate the septa. (See Plate VI., figs. 13 and 14, and Plate VII., fig. 15.)

The subsequent steps in the progress of the disease consist in a further distension of the air-sacs, an enlargement of their perforations, and a rupture of the fibres of which their walls are composed. As these results take place, the walls become more and more imperfect, and the openings in them coalesce. A further breaking down of the walls then occurs, so as to leave but very partial partitions between the cavities; and in the most advanced stages of the disease these partitions undergo other changes, and are reduced to mere membranous shreds, or thin fibrous cords, passing in various directions, traversing, in fact, the distended sacs, two or more of which, by the destruction of their walls, have united to form a single cavity. These cavities occasionally assume a large size, and project from the margin of the lung; they sometimes also form appendages, being connected with the body of the lung merely by stalk-like processes. (See Plate VIII., fig. 16, and Plate IX., fig. 17.)

These appendages differ much in form and volume. In the latter they vary from the size of a nut to that of a pigeon's egg; and occasionally they are even larger. As has been observed by Louis, their elongated and rounded form sometimes makes them resemble the swimming bladder of a fish. They will sometimes empty themselves from a single puncture; at others, two or more punctures are required before they will completely collapse. They present, in their interior, cavities varying in size, and traversed at all angles by thin membranous shreds. At the distal extremity of the appendages these cavities are larger than elsewhere, and they diminish towards the end where the appendages join the lung.

If we examine the inner surface of the appendages I have just described, or of a portion of emphysematous lung-tissue which has reached an ordinary degree of development, we find abundant evidence that the cavities are formed by the dilated air-sacs. We recognise at once, under the microscope, the same appearance of the lining membrane — viz., its epithelial covering — as we find in the air-sacs in a condition of health.

I have observed an anatomical difference in preparations of different lungs, which I think is important in its pathological bearing. In some lungs, in which the emphysema has been of the *lobular* kind, I have found the air-sacs distended to a very considerable extent, but I have observed little or no perforation; whilst in other lungs, and especially in those where the disease has been of the *lobar* character, I have found extensive perforation, with certainly not more, and in some instances less, dilatation than in those alluded to above. In all the cases of lobar emphysema which have come under my notice, I have found the walls of the air-sacs extensively perforated; whilst, in some cases of lobular emphysema, this condition has not existed. These facts would seem to indicate that, in the cases where rupture takes place under a dilatation which, in others, does not produce such rupture, there must be some degeneration of the lung-tissue which renders it more liable to give way.

The condition of the blood-vessels in an emphysematous lung, affords evidence of the cause of its anæmic appearance. We find in the earlier stages of the disease, when there is simply a dilatation of the lung-tissue, that the capillaries of the pulmonary plexus are wider apart than in a state of health; the meshes formed by them are larger. As the walls become perforated, and the sacs further distended and broken, the capillaries become ruptured; and hence we have one cause of the hæmoptysis which occasionally attends the progress of the disease, although this symptom does not necessarily follow from the pathological condition just alluded to.

If we examine a piece of lung in a condition of extreme emphysema, we observe a number of small blood-vessels taking their course in the membranous septa and shreds, which I have previously described as traversing the cavities. The vascularity of these parts is extremely slight; and but little or no respiratory function can be performed by them.

In examining the bronchial tubes in emphysematous lungs, I have found them occasionally dilated, and, more especially so, in old standing cases. Where recent acute, or long-continued chronic bronchitis has existed, the mucous membrane of the large tubes has been red, injected, and somewhat thickened; but I have usually observed that the smaller tubes were pale, and ex-sanguine, although in some instances I have found them filled with muco-purulent matter. I have not seen ulceration present in any case.

If the smaller bronchial tubes of two portions of the same lung be examined, one portion being healthy, and the other emphysematous, the tubes of the former will be found of a darker colour than those of the latter. This results, in part, from the diminished vascularity of the pulmonary tissue in the emphysematous portion, as compared with that of the other, and, in part, from a similar condition of the bronchial mucous membrane.

An alteration of tissue, I have frequently observed, in old met with one instance where this has occurred to any considerable extent. In the case I allude to, a cavity existed as large as a small orange. The substance of the lung from which the pleura was thus stripped was highly emphysematous; and an opening existed at one spot, through which the air had escaped, and found its way beneath the pleura.

Instances of extensive sub-pleural emphysema are, I believe, rare in adults. M. Guillot, in a paper published in the *Archives Générales de Médicine,* for 1853, has detailed a number of such cases in children, who had been, for the most part, the subjects of long-continued hooping-cough. In these cases the pleura was found, after death, raised from the surface in numerous parts of the lung; and in some instances there was emphysema of the cellular tissue of the mediastinum, and even of the neck. M. Guillot seems to me to heve clearly shown that the pathological condition, just described, was a result of the disease from which the patients suffered, and was produced by the severe fits of coughing under which they laboured.

CHAPTER X.

EMPHYSEMA OF THE LUNGS — ITS PATHOLOGY.

Importance of a correct view of the Pathology of the Disease, in reference to Treatment. The Question of Degeneration of Tissue Considered. All cases of Emphysema not to be included under the same head. Distinction between Partial and General Emphysema. Mr. Rainey's View of Fatty Degeneration. Views of Dr. Williams and Dr. Jenner. Investigations of the Author. Constitutional Nature of the Disease: its Hereditary character; etc.

I HAVE, in the previous chapter, traced out the various anatomical changes which take place in the lung-tissue in emphysema. These changes may be briefly described as distension, perforation, atrophy, and breaking up of the walls of the air-sacs. In different parts of one and the same lung, which may present the disease in different stages, the various conditions enumerated may frequently be seen; for instance, towards the margins or borders of the lung, and at its apex—parts which have a greater tendency than others to become affected — the disease may be seen in its most developed form, and "appendages" may exist; whilst away from these spots, and in the deeper parts of the lung, the stage of perforation, or of simple distension, may not have been passed.

The next point which presents itself in connection with our subject, is the consideration of the pathology of the disease, or the nature of the morbid action which results in the structural changes I have previously described. An examination of this question lies at the root of the therapeutics of the affection, for it is obviously impossible that we can scientifically direct our measures towards checking its progress, as long as our knowledge of its pathology is imperfect. Undoubtedly the subject is still involved in much obscurity; and no good can possibly result from any attempt to dogmatize on it.

For the elucidation of the nature of many pathological processes, a knowledge of morbid anatomy alone is often insufficient; and although it may afford indications of the most important character, confirmatory evidence may frequently be derived from other sources, and especially from an examination of the manner in which a disease is influenced by certain modes of treatment. To this point, and to the indications which may be derived with reference to the pathology of emphysema, from the effect produced on it by certain remedial agents, I shall have to recur in speaking of the treatment of the disease.

The great question for consideration, in connection with the pathology of emphysema, is, whether there is any degeneration of tissue either preceding, or attending, the affection; whether, in fact, there is any local or general condition which so interferes with the normal nutrition of the walls of the air-sacs, as to cause their perforation, rupture, and even total destruction. If it could be shown that, either in the elastic fibres, which are so numerous in the walls of the air-sacs, in the basement membrane, or in the capillary blood-vessels, certain changes take place of a degenerative character, a very important step would have been made in reference to our knowledge of this disease.

The first appreciable anatomical change which occurs in the lung-tissue is an increase in the size of the air-sacs. This must necessarily be attended with a loss of elasticity on the part of the elastic fibres, which, on being stretched by mechanical power, are unable to recover themselves. When we find emphysema existing only as a partial affection, and in conjunction with some old-standing disease of the lung, we can readily imagine that it may have been produced by mechanical violence, whilst the tissue affected was in a healthy state; but when we see the disease, even in moderately young persons, creeping insidiously on, and attacking the whole of one or both lungs, without the previous occurrence of long-continued or violent cough, we can scarcely imagine that changes of so extensive a character can result from mechanical violence, unless there exist some morbid condition of the lung-tissue.

My own observations have led me to conclude that all cases of emphysema cannot be included, either with reference to their pathology or determining cause, under the same head. Where the disease is partial, and situated, as is then usually the case, along the margins and at the apex of the lung, and is associated with, or has followed, some other pulmonary affection,—as, for instance, chronic bronchitis, or, in fact, any disease which has been attended with long-standing or violent cough,—I believe the morbid changes may have been brought about mainly by mechanical violence, without there having been any pre-existing affection of the lung-tissue. The general appearance of the lung-substance, in these cases, is very different from that which characterizes the disease to which the name lobar emphysema has been given; the tissue has more the appearance of that of the healthy lung, but it is paler and more anæmic than the latter. It is quite true that in these cases the same anatomical changes—dilatation, perforation, etc.—take place in the progress of the disease, as in the larger and more formidable affection; but these changes necessarily ensue, not only from a rupture of the elastic fibres and basement membrane, but also from the giving way, and absorption, of the capillary blood-vessels—circumstances which lead to imperfect nutrition and consequent atrophy.

Lobar emphysema is a disease which has been but imperfectly treated of by systematic writers, and has not received the attention which its importance deserves. It is true that some of our great pathologists have recognized it as a substantive affection; but, practically, it has not been sufficiently distinguished from the more partial, and less formidable kind of the disease. As I have before remarked, it is occasionally seen in early life; but the most numerous and most marked instances of it that have fallen under my notice have occured in adults. When fully established, it presents symptoms easily recognized, and these will form the subject of future consideration.

That this form of the disease differs very materially in its pathology from the partial emphysema which I have spoken of in a former paragraph, will, I think, be admitted by all who will give a careful attention to the subject. The insidious manner in which the disease sometimes comes on; the almost entire absence of cough frequently observed, as well as of all other symptoms, except a gradually increasing dyspnœa, and, as the patients constantly describe it, a "smothering in the chest;" the occasional rapidity with which the affection progresses; the secondary consequences which ensue; and the general cachexia which often supervenes; — all point to the grave character of the malady, and, I believe, to its constitutional origin in some degeneration of the pulmonary tissue.

But if emphysema be the result of some degeneration of the lung-tissue, it behoves us to inquire what the nature of that degeneration is. On this point a good deal of obscurity still exists. It does not appear that much attention has been given to this part of the subject; and its important bearings on the treatment of the disease have been but little dwelt on.

In a paper which was presented to the Royal Medical and Chirurgical Society of London, and subsequently published in their "Transactions," Mr. Rainey has described the condition of an emphysematous lung, which he seems to have examined with great care. This lung was taken from a subject forty years of age, and the emphysema seems to have been only partial; for the general aspect of the lung, especially in the vicinity of the emphysematous parts, is described as being healthy. A few tubercles, however, existed in some spots. Mr. Rainey found the pulmonary membrane in the emphysematous portions more or less studded with fatty matter; and he has expressed an opinion that this deposit of fat is the precursor of the perforations and subsequent changes which take place in the disease.* As it does not appear that Mr. Rainey has observed this condition of the lung-substance as a general accompaniment of emphysema, but only in the specimen from which he has drawn the chief conclusions referred to in his paper, and as his observations have not been confirmed by subsequent inquiries, the pathological view he has sought to establish—viz., that the disease is the result of fatty degeneration—cannot be considered as settled.

In the Lumleian lectures for 1862, delivered at the Royal College of Physicians of London, Dr. C. J. B. Williams, in speaking of emphysema of the lungs, says: "It is fatty degeneration of the lung-tissue which aids in bringing about the atrophy and rupture of the cells."†

With the exception of the two authors quoted above, no pathologists, as far as I am aware, have supported the opinion that emphysema is produced by, or attended with, fatty degeneration. On the contrary, one of our most accurate observers of this disease, Dr. Jenner, to whom we are indebted for an able paper on it, has expressed his opinion that "the most frequent anatomical change in the lung," producing loss of its elasticity, "is fibrous degeneration—the consequence of the exudation of that variety of lymph which escapes from the capillaries, when they are the seat of slight but long-continued congestion." *

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* Medico-Chirurgical Transactions, vol. xxxi.

† Lumleian Lectures: *The Lancet*, 1862.

With the view of ascertaining whether emphysema is preceded by, or has associated with it, fatty degeneration of the pulmonary tissue, I have made a careful examination of a large number of specimens of lungs which were the seat of the disease. These specimens were taken from lungs which presented the affection in all its varieties, whether partial or general. I have not only submitted to examination the diseased portions, but (where the disease was partial) pieces taken from contiguous parts, and where the lung-tissue was apparently healthy. The general results of my investigations may be briefly stated as follows: In the large majority of cases I have found no indications whatever of fatty matter; in some few instances, however, I have seen deposits of fat in the walls of the air-sacs.

My examinations have been conducted with the microscope on recent, and dried specimens; and also by heating the lung-tissue between pieces of glass, so as to dissolve out the fat, if present, and thus get indications of its existence.

In examining this question, I have viewed it in several lights; and, considering that the disease might possibly be due to some affection originally commencing in the capillary blood-vessels, and producing mal-nutrition in the pulmonary tissue, I have made a careful examination of the branches of the pulmonary artery, from their commencement to their termination in the pulmonary plexus, in order to ascertain if they were the seat of fatty or atheromatous degeneration. The results are that I have found in some cases

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* Medico-Chirurgical Transactions, vol. xl.

atheroma existing in the branches of the pulmonary arteries, and in their capillaries, whilst in others I have found no indications of it whatever. In the cases where the atheroma existed in the pulmonary arteries, I have always found it in the aorta: so that the affection of the former vessels must be considered as simply the result of the general tendency to arterial degeneration, and not as possessing any specific bearings on the emphysematous condition of the lung-tissue.

I have further endeavoured to ascertain, by careful microscopical examination, whether any appreciable difference could be traced between the elastic fibres of the emphysematous lung, and those of the healthy lung. Here, again, I am not able to say that any marked distinction existed between the diseased and healthy fibres, except that the former sometimes appeared less regular in their outline, and had less tendency to curl up at their ends; but as to structural change in the fibres, I have not been able to satisfy myself that any had taken place.

Considering, therefore, the facts and statements I have adduced in connection with this question, I cannot agree with the view that has been expressed as to the dependence of emphysema on fatty degeneration; for when we find that this condition is only an occasional, and not a constant, accompaniment of the disease, we cannot look upon it as its essential and predisposing cause. Nor can I, on the other hand, agree with Dr. Jenner in his view of the nature of the degeneration—at least as regards the lobar form of emphysema. It is quite possible that, in cases of chronic bronchitis, there may be congestion of the pulmonary plexus, which may give rise to a weakening of the walls of the air-sacs, and that, under this condition, the sacs may become distended and ruptured by the act of coughing; but this will not apply to those cases where the emphysema is of the primary kind, coming on without any pre-existent affection of the bronchial tubes. In such cases the degenerative process is the first step in the disease; and any congestion which may occur is but a secondary consequence.

But although microscopical examination does not enable us to detect any structural alteration in the ultimate tissues of the air-sacs, and the application of other means furnishes us with no proof of the presence in them of any morbid condition; yet, that degeneration does not exist is by no means determined by the failure of our present methods of investigation to demonstrate it. It is quite possible that the elasticity of the yellow fibres may become impaired, or even destroyed, without any structural alteration resulting, such as could be appreciated even with the highest powers of the microscope; and it is also equally possible that changes may occur in the blood-vessels, giving rise to mal-nutrition of the pulmonary tissue, and yet, that we may be unable to distinguish them. We see in this disease, as in many others. the secondary changes, as I have described them in the previous chapter; but the primary and essential ones we cannot recognize.

Notwithstanding, however, that my investigations do not enable me to say what is the exact nature of the degeneration which leads to the production of emphysema, nor yet, whether it commences as an affection of the capillary blood-vessels, or of the elastic fibres and basement membrane, I do not entertain the slightest doubt that *the disease in its severer forms is of a constitutional nature; that one of its most important features, and perhaps the primary step in it, is a mal-nutrition of the pulmonary tissue, causing its degeneration, and giving rise to all the structural changes which I have previously described.* That fatty deposit occasionally exists, I have already stated;

but the question arises whether this is a primary cause of the anatomical changes which take place, or whether it may not be the result of the imperfect nutrition which necessarily ensues in the progress of the disease.

The view which I have taken, of the constitutional nature of emphysema, receives support from the facts which have been brought forward with reference to its hereditary character. On this point I quote the observations of Jackson, which furnish us with very important results. He found that, of twenty-eight persons suffering from emphysema of the lungs, eighteen were born of parents (father or mother) affected with the same disease, several of whom had died of it. In some instances the brothers and sisters of these persons were also emphysematous. On the other hand, of fifty persons not affected with emphysema of the lungs, three only were born of emphysematous parents.

Facts of this kind tend to throw great doubt on the opinion that emphysema is solely produced by mechanical dilatation of the healthy air-sacs, and to favour the view that it has some deep-seated pathological cause in connection with the lung tissue.

Some pathologists have supposed that there is hypertrophy of the pulmonary tissue in emphysema, and M. Louis has endeavoured to account for this condition, on the principle that all membranous structures become thickened in proportion as they are dilated; and he quotes, as instances, the effects produced on the œsophagus by cancer of the cardiac orifice of the stomach —on the stomach by a like disease of the pylorus— and on the walls of the heart by dilatation of that organ in consequence of obstruction. My own researches do not enable me to agree with the view that any real hypertrophy takes place in emphysema; and with reference to the explanation which has been given by M. Louis of the causes producing hypertrophy, it appears to me that no analogy whatever exists between the cases he has brought forward and the disease under consideration. It is true that in emphysema there is a retention of air in the air-sacs, but there is no obstruction to its exit from them; and even if such obstruction did exist, there would be no resemblance between the condition thus produced in the lung, and that produced in the organs alluded to by M. Louis. The air is not driven from the air-sacs—as the blood is driven from the heart, or the food from the stomach—by forcible contraction of their walls, but by their elastic reaction. Now, in emphysema this elasticity is impaired, or lost; the air is consequently retained passively, and not because it cannot be forced through an obstructed orifice. The air-sacs are not called upon to perform extra work; and no element of hypertrophy, such as M. Louis supposes, can consequently exist.

In considering the pathology of a disease, as I have before remarked, we often derive material aid from observing the manner in which it is influenced by certain remedial agents; and with reference to emphysema, I think we may gather from this source very important indications. From a close study of the disease for some years past, and a careful observation of the results of treatment in numerous cases, I am convinced that, setting aside the bronchial and asthmatic symptoms which are so constantly associated with the affection, the main principles on which emphysema should be treated are precisely those which guide us in the treatment of diseases attended with degeneration: such, for instance, as Bright's disease of the kidneys and fatty degeneration of the heart. The principles must be modified in reference to the peculiarities of emphysema; but, as all these points will form the subject of consideration in the chapter devoted to the question of treatment, they are merely alluded to here in connection with certain inferences which will be found below.

In concluding this portion of my subject, I shall state briefly the circumstances which induce me to believe that emphysema is the result of some degenerative process; and although it would be more satisfactory to be able to speak positively as to the exact nature of the degeneration, yet, for all practical purposes, it is sufficient to point out the general pathology of the malady, and to indicate the principles on which it should be treated.

1st. The high degree of development which the disease often reaches, without any previous history of violent or long-standing cough, either in connection with bronchitis, hooping-cough, or any similar affection.

2nd. The frequency with which the disease attacks the whole of both lungs; and the uniformly equal character of the morbid changes often observed throughout all parts of the lungs.

3rd. The hereditary nature of the disease, as shown by the observations I have alluded to.

4th. The manner in which the disease is influenced by certain remedial measures which are known to act beneficially on other diseases attended with degeneration of tissue.

CHAPTER XI.

EMPHYSEMA OF THE LUNGS—ITS DETERMINING CAUSES AND MECHANISM.

Two Principal Theories of the Determining Causes of the Disease. Views of Laennec. Theory of Dr Gairdner. The Mechanism of Respiration. Effects produced by Collapse of the Lung. The Expiratory Theory. The Modus Operandi of Expiratory Efforts. The Anatomical Arrangement of the Walls of the Chest, and the Disposition of the Lungs. Effects of Forced Expiration. The case of M. Groux. Results of the Author's Observations. Infrequency of the Disease as a Sequel of Pleurisy and Pneumonia; its frequent occurrence in Tubercular Lungs. Cases recorded by M. Guillot.

I HAVE endeavoured to show, in speaking of the pathology of emphysema, that all cases cannot be included under the same head, and that some, in all probability, arise spontaneously; whilst others may be dependent upon a pre-existent affection of the bronchial tubes, and may be caused by mechanical violence. The connection of the disease with bronchitis has long been recognized by pathologists; and, with few exceptions, the general opinion seems to have been that this alone was its determining cause. It is true that Lebert admits the spontaneous origin of emphysema; and that Hasse, whilst agreeing with the general opinion that catarrh is the principal occasional cause of the disease, states that its character and real import depend on other causes, not thoroughly made out. Louis also, in his "Researches on Emphysema," denies the dependence of the disease in all cases on pulmonary catarrh; and admits the possibility of its development without any such previous affection. The observations of Louis are so important on this point, and the statement he makes, as to the existence of dyspnœa and oppression long before the occurrence of cough, so entirely accord with the results of my own experience, that I quote the following remarks from his paper:—"Setting aside the cases where dyspnœa could be traced back to very early youth, and in which cough did not generally supervene till much later, the oppression was not nearly always preceded by pulmonary catarrh; and in many patients catarrh did not come on till one or more years after the commencement of the oppression; whence this necessary conclusion—that emphysema can develop itself, and in fact does tolerably frequently develop itself, without pulmonary catarrh. This conclusion is again, in some measure, confirmed by this further fact, that dyspnœa often appears not to have increased in an appreciable degree after a severe acute pulmonary catarrh; and when we recollect that the maximum of emphysema usually has its seat along the thin borders of the lungs and their adjacent parts, whilst acute pulmonary catarrh has its seat behind and below, we shall be forced to conclude that, if catarrh has any influence whatever on the development of emphysema, that influence is slight, and, without doubt, but rarely exercised." *

I think Louis has under-estimated the influence of bronchial affections in giving rise to emphysema; but of the correctness of his observations with reference to the early existence of dyspnœa I have had abundant proof.

Although there is now, for the most part, a general agreement amongst pathologists, as to the dependence of emphysema on bronchitis — and as far as regards the partial forms of the disease I entirely agree with the view — and

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* Recherches sur l'Emphysème des Poumons. Mémoires de la Sociéte Médicale d'Observation. Paris, 1837. Tome i., p. 160.

further, that its mechanical production is in some way associated with bronchial inflammation, yet, the exact manner, in which the dilatation and rupture of the pulmonary tissue are brought about, has been the subject of much discussion, and has given rise to great diversity of opinion. As the question is one not only of great interest, but of much practical importance, it is necessary to consider it at some length.

Speaking generally with reference to the determining causes of emphysema, we may say that two principal theories have been entertained: these are, respectively, called the inspiratory, and the expiratory, theory.

By those who hold the former view, it is supposed that dilatation and subsequent rupture of the air-sacs of the lung take place as the result of their over-distension during an inspiratory act; by those who entertain the latter view, that these results are brought about by expiratory efforts, especially by the act of coughing.

Laennec, recognising the frequency of the disease as a sequence of pulmonary catarrh, supposed that it was occasioned by an over-distension of the air-cells, from an accumulation of air taking place in them, in consequence of the obstructed condition of the bronchial tubes. He says:—"The small bronchial tubes are distended by the viscid mucus, or by the swelling of the mucous membrane. Now, as the muscles which act in inspiration are strong and numerous, and as expiration, on the contrary, is only produced by the elasticity of the parts and the feeble contraction of the intercostal muscles, it must often happen that in inspiration, the air, after having overcome the resistance which was opposed to it by the mucus, or by the tumefaction of the mucous membrane, cannot overcome it during expiration, and remains imprisoned. The following inspirations add further to the dilatation of the cells to which the obliterated tube leads. Lastly, the distension, by the heat of the lungs, of the air introduced cold into the chest, must contribute to this dilatation."

The theory, thus advanced by Laennec, is based on a view of the respiratory function which has been proved to be essentially incorrect; viz., that the inspiratory power is greater than the expiratory. The researches of Hutchinson and others have shown that the power of forced expiration considerably exceeds that of inspiration. This important physiological fact cannot be too constantly borne in mind, in considering the nature of emphysema.

But further, it has been shown by the researches of Gairdner, and others, that an accumulation of mucus in the bronchial tubes, such as Laennec thought would lead to a distension of the air-sacs, has an exactly opposite effect; and is, in fact, followed by a collapse, and not a dilatation, of the pulmonary tissue. Dr. Gairdner has shown that the pathological condition of the lung, which has been known under the name of lobular pneumonia, is nothing more than a collapse of the lung tissue, which — unless the affection have existed for a long time, and have produced atrophy — may be readily distended, by inflation through the bronchial tube leading to it. He has found this state of the lung following bronchitis, and constantly associated with obstruction of the bronchial tubes leading to the affected part. He accounts for the production of the collapse in the following manner: —

"The bronchi are a series of gradually diminishing cylinders, dividing for the most part dichotomously. If a plug of any kind, but especially one closely adapted to the form of the tube, and possessing considerable tenacity, be lodged in any portion of such cylinder, it will move with much more difficulty towards the smaller end, and in doing so will close up the tapering tube much more tightly against the passage of air, than when moved, in the opposite direction, into a wider space. If such a plug be placed over a bifurcation, it will, even if freely moving in the larger space in which it lies, be of sufficient bulk to fall back upon one or other of the subdivisions during inspiration, in the manner of a ball-valve upon the orifice of a syringe, and thus completely occlude it. The consequence of this mechanical arrangement must inevitably be, that at every expiration a portion of air will be expelled, which, in inspiration, is not restored, partly owing to the comparative weakness of the inspiratory force, and in part to the valvular action of the plug. If cough supervene, the plug may be entirely dislodged from its position, or expectorated, the air, of course, returning freely into the obstructed part; but if the expiratory force is only sufficient slightly to displace the plug, so as to allow of the outward passage of air, the inspiration will again bring it back to its former position; and the repetition of this process must, after a time, end in a perfect collapse of the portion of lung usually fed with air by the obstructed bronchus."*

In confirmation of the view, that obstruction of the bronchial tubes leads to collapse of the pulmonary tissue, we have the results of experiments performed by Mendelsohn and Traube. These experimenters introduced into the trachea of certain animals small hard bodies, which they pushed down into the bronchial tubes as far as possible. The general result found, on examining the lungs of these animals after death, was, that the portion of lung connected with the tube which was obstructed by the introduced plug was red and void of air; in fact, in a state of collapse. In addition to these facts and observations, which may be adduced against the theory advanced by Laennec, it may be stated that the seat of pulmonary emphysema, when it exists as a partial affection, and that of obstructed bronchial tubes, is not the same; in fact, the two affections have altogether different localities, bronchial inflammation and collapse being most frequent in the lower and posterior parts of the lungs, and emphysema in the upper and anterior parts. It has been shown, by the researches of Gairdner, Lebert, and others, that pulmonary collapse and emphysema are frequently found existing together in the same lung; and the former author has so constantly seen the two affections associated together, that he has looked upon them as having the relation to each other of cause and effect. Hence he has sought for an explanation of the production of emphysema, in the altered relation which the collapsed lung bears to the cavity in which it is placed, as compared with its relations in a state of health. His opinions may be summed up as follows:—Adopting the view that emphysema is produced by the force of the inspired air acting on the walls of the air-sacs, he considers the disease in the light of a complementary lesion, depending upon the fact that a portion of the lung has become *diminished in bulk and incapable of distension.* In consequence of this condition of the lung, which is found in pulmonary collapse, those portions of the organ which remain in a sound state receive into them a larger quantity of air than usual, in order to fill the space previously occupied by the portion now collapsed: hence over-distension of the air-sacs and rupture.

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* The Monthly Journal of Medical Science, vol. xi., p. 242.

Notwithstanding the very able manner in which the author of the above theory has supported his views, it appears to me that there are circumstances which tend to invalidate his conclusions; and that the exclusive doctrine of the production of emphysema which he has advocated will not bear the test of strict clinical investigation. That, when one portion of a lung is collapsed, it necessarily follows that the sound portions of the organ will expand beyond their usual size, so as to fill the space previously occupied by the collapsed lung, and dilate the thoracic cavity to the same extent as before, appears to me to be opposed to what we know of the mechanism of respiration. During respiration the chest expands, to make room for the dilating lungs, and it will only expand to the extent required by the amount of air which enters the lungs. The air is drawn equally to all parts of the lungs; neither the muscles of the chest, nor the lungs themselves, have any power to *determine* the air to one part of the organs more than to another. No external force exists which can accomplish such a result; and therefore it seems difficult to understand how a diversity of currents could be produced in different portions of the lungs, so great as to lead to an over-distension of some parts, whilst others remained normally dilated. That, when collapse of a portion of the lung takes place, so that no air can be received into it, the same quantity of air as previously entered finds its way into the chest, so as to dilate it to the same extent as before, appears to me extremely doubtful; and such a view is, in my opinion, opposed to other pathological facts which we witness in connection with the lungs. But if we admit that this view is correct, and that in proportion as some parts of the lung collapse, others become more than normally dilated, so that the chest reaches its previous state of expansion, then the air would become diffused throughout the whole of the lung remaining sound, and not be driven, or drawn, to any particular locality. The consequence of this would be, that an increased small dilatation of every part would compensate for the want of action of the small collapsed portion. There would, under these circumstances, be no special strain on any particular part of the pulmonary tissue—no rush of air to one part more than to another. But further; if, as the result of pulmonary collapse, any portions of the lung become abnormally distended, so as to lead to the production of emphysema, it appears to me that it ought to be those which lie in contiguity to the collapsed tissue. But we do not find this to be the case; on the contrary, the collapsed portions are most frequent in the posterior and lower parts of the lungs, the emphysematous at the apex and along the margins. It is true that Dr. Gairdner has found, in some cases, patches of emphysema lying side by side, with the collapsed tissue, and I have seen the same thing myself; but, as a rule, the two affections have, as I before stated, different seats.

From the views I have expressed above, it appears to me very difficult to account for the production of emphysema by a forcible distension of the lung as the result of an inspiratory act; and, especially, if we consider the amount of distension which the lungs will bear, when in a healthy condition, without any rupture of the air-sacs taking place; a distension, probably, far greater than they undergo in those cases of disease where one lung, or part of one lung, takes on increased action, to compensate for the want of action of a disabled portion.

I pass now to consider briefly the expiratory theory of the disease. Are there any circumstances in ordinary or forced expiration which have a tendency to produce a distension of any parts of the lungs, and consequently to lead to the production of emphysema? With reference to the act of ordinary expiration, we may safely say that there are none; but in regard to forced expiration, it appears to me that such circumstances do exist.

It has been urged, as an objection to the theory we are now considering, that the expiratory act is mechanically incapable of producing distension of any part of the lung; and that the air-sacs are emptied by an uniform pressure of the thoracic walls upon the whole pulmonary surface. This objection only applies to the ordinary act of expiration; and it is undoubtedly true that the lungs then undergo equable pressure on all parts, and that there is no tendency for the air to be forced towards, or retained in, any particular part of the pulmonary substance; but the argument loses its weight when we come to apply it to forced expiration, as, for instance, to the act of coughing. The effect which is produced on the lung by coughing has been pointed out by Dr. Jenner, in a paper read before the Royal Medical and Chirurgical Society of London, and already referred to; and the remarks he has there made entirely accord with what I have myself observed. If we examine a person whose chest is exposed during the act of coughing, we see a distinct bulging produced above the level of the clavicles; in fact, it is clearly shown that the air is forced upwards by this expiratory act, and that it forcibly distends the upper parts of the lungs. Percussion of the tumour, formed as I have stated, yields a resonant sound, which becomes almost tympanitic if the lung be in an emphysematous state. Now, if we examine the anatomical arrangements of the walls of the chest, we have a ready explanation of the phenomena to which I have just alluded. The lateral and inferior walls of the thoracic cavity are strong and resisting, and by their elasticity they assist actively in expiration; further, the contraction of the abdominal muscles forces upwards the diaphragm, especially in violent expiration. The part of the thoracic walls which is the weakest, and which offers the least resistance, is that which separates the cavity of the chest from the region of the neck. We there find a fibrous structure, a strong fascia, in fact, connected externally with the first rib, and internally blending with the cervical fascia as it passes down into the chest. This plays no active part in the expiratory process, and offers no active resistance to the distension of the lung. From a knowledge of this peculiar arrangement of the walls of the chest, and a consideration of the action of those muscles which are concerned in expiration, it appears to me that, during violent expiratory efforts, the lungs must be unequally compressed, and that air must be driven, first, to those parts of the lungs where the walls are least resisting; and, secondly, to those portions which contain the least volume of air.

I have shown above that the apices of the lungs are the parts covered by the least resisting walls; and it will at once occur to all, that the parts which contain the least volume of air are the anterior borders and the margins of each base. These parts are not only the thinnest, but they are also out of the direct line of strongest pressure which the lungs undergo in expiration. Violent expiratory efforts are chiefly made with the abdominal muscles, and the most powerful agents are the recti; the contraction of these muscles, forcing upwards the abdominal viscera and the diaphragm, produces the greatest amount of compression at the base of each lung; the air is consequently driven upwards in a strong current. There being no corresponding force acting at the upper part of the chest, on the apex of the lung, this latter is not emptied; on the contrary, it becomes forcibly distended by the upward current. Further, the strong currents of air from the central and basic portions of the lungs overcome those from the thin portions; and thus these latter, instead of being emptied, become like the apex, forcibly distended.

Dr. Jenner supposes that the cartilaginous portions of the thoracic walls are somewhat yielding, and thus accounts for the production of emphysema along the borders of the lung. This explanation seems to me doubtful, and the one I have given far more probable.

Again, I may refer to the phenomena which were witnessed in the case of M. Groux, who was over in this country some years ago, and made a tour of many of the metropolitan and provincial towns. In this case a fissure of the sternum existed, which allowed of some of the movements of the heart being observed. Those who examined M. Groux will recollect that, during a violent expiratory act, the lung of one side came forward in the upper part of the fissure, and formed a distinct, elongated, resonant tumour; no such result taking place during inspiration. Whatever influence this fact may have on our minds with reference to the expiratory theory of emphysema, it tends, at any rate, to show that, wherever there is a weak part, or an absence of compressing power, in the thoracic walls, the lungs will there undergo distension during forced expiration.

The facts and arguments I have adduced seem to me to prove that the objection to the expiratory theory, on the ground that the expiratory act is mechanically incapable of producing distension of any part of the lung, is untenable; and without expressing my own attachment to any exclusive theory of the production of emphysema, I may remark that my observations of a considerable number of cases have led me to conclude, that by far the most frequent cause of the partial form of the disease is to be found in the cough attendant on bronchitis, or some other affection of the bronchial tubes, such as pertussis. The fits of coughing caused by the repeated attacks of bronchial inflammation must, it appears to me, so react on the pulmonary vesicles which are most liable to distension, as to produce, after a time, their dilatation and rupture; and I the more incline to this view of the production of the disease, from knowing how difficult it is to produce artificial emphysema by inflation of the lungs, even after death. It may be objected to this view, that emphysema should be more frequently found as a sequence of pleurisy or pneumonia. We know that in some cases of inflammation of the lungs partial emphysema *is* produced. I have seen this when the emphysema was apparently quite recent, and had, probably, been produced during the progress of the pneumonia. Such a result, however, only rarely follows, and I think we have an explanation of the fact in the character and short duration of the cough attendant on this disease, as well as on pleurisy.

Again, it appears to me that the frequency with which we meet with emphysema in tubercular lungs, favours the view I am taking. The deposition of tubercles in the pulmonary tissue filling up the air-sacs in the same way as a pneumonic exudation, with the subsequent gradual contraction of the walls of the chest, presents no condition favourable to the formation of emphysema; except the cough, which is usually so important a feature of the disease.

M. Guillot has collected a series of cases, to which I have already referred, illustrating the effects of long-continued spasmodic cough in producing emphysema. These cases, recorded with some excellent observations, are fifteen in number; the subjects of them were infants, most of whom suffered from pertussis, but in all, long-continued spasmodic cough was a very prominent symptom. Death took place in all, and the *post-mortem* examination revealed the existence, to a very considerable extent in some cases, of sub-pleural emphysema, with, in some instances, extra-

It will be seen that, in advocating this view, I am, all the while, supposing that the primary step in the disease is a degeneration of the lung-tissue, and that the mechanical distension is a secondary consequence.

If the views I have enunciated be correct, it follows that not only must we place different kinds of emphysema under different heads as to their pathology, but also as to their determining cause.

CHAPTER XII.

EMPHYSEMA OF THE LUNGS — ITS SYMPTOMS AND PHYSICAL SIGNS.

Dyspnœa and Cough. Hæmoptysis. The Occurrence of Asthmatic Symptoms. Aspect of Patients. General Configuration of the Chest. Movements of the Chest in Respiration; their Variations in different Cases. Causes of these Variations. Character of Inspiration and Expiration, and of the Act of Coughing. Percussion and Auscultation. Character of the Respiratory Sounds. The Physical Signs and Symptoms referable to the Structural Alteration in the Lung-Tissue. Diminution of the Respiratory Function, and of the Quantity of Blood circulating in the Lungs. Diminished Temperature of the Body.

Amongst the most important, and most frequent symptoms of emphysema, is a constant, and, generally, increasing shortness of breath. It will usually be found that the patient has been for some time subject to this symptom, which is always increased on exertion, especially walking up-hill or up-stairs, on the occurrence of a catarrhal attack, or whenever the stomach is much distended. The dyspnœa is relieved during the summer months, but returns again, with increased severity, with each succeeding winter. Cough is, generally, more or less present, and is accompanied by expectoration in proportion to the bronchitic symptoms. The expectoration varies in its character, and is, occasionally, streaked with a small quantily of blood. Hæmoptysis, however, never exists to any great degree: its occurrence is undoubtedly due to the rupture of the capillary blood-vessels, which takes place as the lung-tissue is distended. Such rupture must frequently occur, but in the majority of cases it is unattended with hæmorrhage. The patient usually complains of no pain, but of a general feeling of oppression, or "smothering in the chest," as he often terms it. It frequently happens, in the most severe cases of lobar emphysema, that this last symptom, and the increasing dyspnœa, at times becoming very urgent, are the only circumstances which have attracted the attention of the sufferer to his disease. In other cases, however, and especially where the emphysema is only partial, a close examination will elicit the fact that bronchitic attacks have preceded the affection, and have been associated with it in its progress.

Few, if any, cases of emphysema, especially where the disease is extensive, exist for any lengthened period without the occurrence of asthmatic symptoms. The patients complain of being attacked with difficulty of breathing, and tightness across the chest in the night. The attack usually comes on some time between twelve p. m. and four a. m., and, after lasting a variable time, subsides in a fit of coughing, attended with expectoration. As I shall have, hereafter, to speak of the close relation existing between emphysema and asthma, I shall not dwell on the subject now.

In advanced cases of the disease, the aspect is peculiar, and very characteristic; the countenance is dusky, and sometimes has a puffy appearance, the result of venous congestion, and indicative of the imperfect manner in which the aeration of the blood is performed. The nostrils are dilated, and are seen to expand widely at each inspiration; at the same time the angles of the mouth are drawn downwards. The voice is feeble, and there is an inability to sustain a prolonged, uninterrupted expression. The whole body has more or less of a cachectic appearance, and is sometimes much wasted. To these symptoms must be added those of general dropsy, which not unfrequently follows as a consequence of the effects produced by the disease on the right cavities of the heart.

Amongst the most important of the physical signs of emphysema may be enumerated the following:—The upper part of the chest and the clavicles are very prominent; the neck appears shortened; in fact, the whole of the thorax is drawn upwards, for the ribs have in great measure lost their obliquity, and their anterior extremities are at a higher level than in a state of health. The fossæ above the clavicles are deepened,* and the tense cords of the hypertrophied sterno-cleido and scaleni muscles stand out in relief. There is increased curvature of the dorsal spine, and the sternum becomes arched instead of straight. The gait is stooping, and the head and shoulders are thrown forwards. When in bed, the patients will often be observed to sit up, with the body bent forwards, and the arms folded and resting on something, in order that the accessory muscles of inspiration may be the more readily brought into play. The ribs are prominent, and the intercostal spaces, generally, appear depressed. In well-marked and advanced cases, there is an increase in the size of the chest, and a rounded or convex condition, especially of its upper part. The existence of an enlarged thorax in emphysema has been maintained by almost all those who have written on the disease; but the fact has been disputed by Dr. W. T. Gairdner, who asserts that there is no enlargement of the chest as a whole, and that the increased capacity of the upper, is counteracted by a diminution of the lower, part. I am disposed to think that Dr. Gairdner is in error in regard to this point, and that he has been misled by the fact that, in emphysema, there is always more movement of the upper costal portion of the chest than of the lower. Indeed the lower ribs scarcely move at all; and in bad cases, what movement of them does take place is just the opposite of that which occurs in health, for, together with the lower end of the sternum, they fall in during inspiration. My belief with regard to this question, of increased capacity of the chest, is, that there exists, for the most part, a considerable enlargement of the upper part, and no diminution of the lower. There are some cases, however, in which the floor of the chest yields greatly to the expanding lungs, and where, consequently, from the diaphragm being pushed down, the thorax acquires increased depth; hence the dilatation of its upper part is less marked.

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* M. Louis speaks of a prominence behind the clavicles in some cases of emphysema, by which an appearance of plumpness was given to the supra-clavicular regions. I cannot say that I have observed such a condition in well-marked cases that have come under my notice; and I am confirmed in the remarks I have made in the text, as to the existence of a depression behind the clavicles, by the testimony of Hasse, who says he has never observed anything like a prominence. The depression alluded to is produced, not by the falling away of the lung, but by the elevation of the clavicles.

The conditions I have described are those which are found when the disease is extensive. When it is partial, confined, for instance, to one lung, or to parts of one lung, the prominence of the chest may exist on one side only, and the other symptoms enumerated will be less marked.

The movements of the chest in respiration are peculiar. The breathing is for the most part superior thoracic; there is, however, not much dilatation of the chest, even at its upper part; for the distended lungs having already inordinately expanded the cavity, there is but little room for further enlargement. The accessory muscles of inspiration in the neck, the sterno-cleido mastoid and the scaleni, are seen in constant, and often powerful, action. The chest is raised, and slightly pushed forwards above, during inspiration, whilst below, it sinks in. This recession of the lower part of the sternum, and of the lower ribs, is very obvious in well-marked cases of the disease.

The respiratory movements vary in different cases, depending on causes which I shall explain hereafter. In some patients we find that there is little or no action of the diaphragm in inspiration; in fact, during this process the abdomen remains flattened, or even concave. Here the upper part of the chest has yielded to the expanding lungs, and is very prominent. In a second class of cases the respiration is strikingly different. At every inspiration the thorax is raised, almost straight upwards, with even less expansion than is observed in the first set of cases, whilst at the same time the abdomen is protruded, somewhat forcibly and rapidly. The abdominal protrusion differs from that which takes place in a forced inspiration in health, in that the part protruded is lower; for, whilst the lower abdominal regions are pushed forwards, the upper ones, together with the lower ribs, are drawn inwards.

The different character of the breathing, in these two classes of cases, was accurately pointed out by Dr. Stokes; and I can fully confirm the observations he has made as to the facts I have referred to.

Upon what causes do these differences in the respiration depend? It is obvious, I think, that they must be explained on the ground that, in the one set of cases, the diaphragm has been pushed downwards by the expanding lung, whilst, in the other, it has maintained its normal position, and the principal yielding has been in the upper part of the thorax. From my own observations, I am disposed to believe that the cases, which are not characterized by displacement of the diaphragm, are those where the disease is chiefly located in, or has originally attacked, the upper part of the lungs. Here the bony walls of the chest have yielded before the expanding organs. Where, however, there is diaphragmatic displacement, I believe it will, usually, be found that the disease is of a general character, that the whole of the lungs is affected, and that the malady has commenced and progressed in their lower, at the same time as in their upper, parts. I am supported in this view by the fact that these cases are the most urgent in their symptoms, and are characterized by the greatest amount of dyspnœa. I have had opportunities of examining such cases after death, and have found the lungs universally emphysematous, their bases flattened, and the diaphragm, with the abdominal viscera, displaced downwards.

The respiration presents another feature, which, in well-marked cases, is alone sufficient to indicate the nature of the disease. The inspiration is short, and quick, and is followed by a prolonged, and often wheezing, expiration. The former phenomenon is due to the small expansion which the chest undergoes, and the quick action of the inspiratory muscles; the latter, to the slowness with which collapse of the lungs takes place, in consequence of their loss of elasticity.

The act of coughing, in emphysema, is always feebly performed, and expectoration is attended with difficulty. This is of more serious importance, in consequence of the great liability of emphysematous patients to bronchitis, and of the profuse secretion which occurs in such attacks, if at all acute. The presence of mucus in the bronchial tubes causes irritation, which is only increased if the attempts to raise the secretion are ineffectual; and thus spasmodic fits of coughing are often produced, and even suffocation is occasionally threatened.

Percussion and auscultation elicit important diagnostic marks in this disease. Where the affection is general, there is increased, and, in some instances, almost tympanitic, resonance over the whole of the chest, most marked towards the apices of the lungs, and along their anterior borders, and, in partial cases, almost confined to these spots, or to one side, if only one lung be affected. Unlike what occurs in healthy lungs, viz., that the clearness of the percussion note is augmented after inspiration, this latter act produces but little change in the resonance of the emphysematous organ. In advanced cases, and especially where both lungs are affected, the præcordial region in resonant, from the heart having been pushed backwards, downwards, and towards the median line, by the expanding and overlapping left lung. In some partial cases the cardiac region retains almost its normal amount of dulness; and again, in other cases, from pleural adhesions having prevented the lung from overlapping the heart, the latter organ may be in its usual position as regards the walls of the chest. Again, the enlargement of the right side of the heart, which so frequently follows in this disease, may sometimes, but I think very rarely, give rise to dulness on the right side of the lower part of the sternum.

An examination of the back of the chest will show that the line of resonance has a lower level than in health. In the normal state, the lungs do not reach to the bottom of the pleuræ; but, when enlarged by emphysema, their lower margins are pushed down to the extreme base of these cavities. The cardiac impulse can be neither seen, nor felt, in the normal position; in advanced cases, however, from the expanding lung having dislodged, and encroached on the space of, the heart, the latter is observed beating, just below the ensiform cartilage, in the epigastric region. The impulse there is strong, and offers a marked contrast to the pulse, which is generally small and feeble. These phenomena are due to certain changes which take place in the heart, and which will form the subject of future consideration.

I have spoken of the displacement which the heart undergoes in emphysema. There is another circumstance to which I must refer, in connection with its sounds. Generally speaking, when listened for, at the usual position of the apex, the sounds are fainter and more obscure than natural; but very frequently, if the examination be made at a lower level, and nearer the median line, they will be distinctly heard. In fact, in consequence of the displacement of the heart, the site at which the sounds are best heard is altered. This remark especially applies to those cases where the heart can be felt beating in the epigastrium, and where, consequently, the displacement is considerable; in other cases, where the heart is merely pushed back and overlapped by the lung, the sounds are everywhere faint and obscure.

The quantity of air, entering and leaving the lungs in emphysema, is small; hence the respiratory murmur is faint, and characterized by peculiarities which a knowledge of the anatomical condition of the lung-tissue, and of the walls of the chest, at once enables us to explain. The inspiratory sound is short, sometimes very faint, and is followed by a prolonged expiratory murmur. This latter is unlike the sound heard in any other affection, and is, in fact, pathognomonic of emphysema.

In the early stages of the malady, the respiratory sounds are merely faint, and no marked prolongation of the expiratory murmur is heard. It is important to be aware of this fact, and to recognize the affection at an early period of its course, when there is the greatest probability of the use of remedial agents being attended with success. Auscultation may here give more valuable aid than percussion, for some dilatation of the air-sacs may exist, without causing that amount of resonance which would attract attention; in such cases, however, the feeble character of the breathing will clear up any doubt we may entertain.

In some advanced cases of the disease, the respiratory sounds are scarcely audible, if the bronchial tubes are free from inflammatory action, and no spasm exist.

Laennec has described a *râle* which he considered peculiar to, and pathognomonic of, pulmonary emphysema. He calls it, *râle crépitant sec à grosses bulles.* Most authors who have written on emphysema since Laennec, have expressed doubts as to the occurrence of any *râle* characteristic of the disease. I have, on several occasions, heard a *râle* in emphysema, which I believe to be similar to that described by Laennec, and I have never had the slightest doubt as to the nature of the cases in which it occurred. The diagnosis, however, was not formed simply from the existence of the *râle*, but from the presence of other more prominent symptoms. Laennec described the *râle* as *dry*, and supposed that it was produced by the distension of the emphysematous lung-tissue. The *râle* with which I am familiar, appears to me of a moist, rather than of a dry, character, and to be closely allied to the sub-crepitant *râle* of bronchitis. In many cases of extensive emphysema it is not heard, and in the instances in which I have found it, there has been previous inflammation of the bronchial tubes. My belief is, that the *râle* is produced, not in the way that Laennec supposed, but in consequence of the presence of a certain amount of fluid in the finer air-passages. As a sign of emphysema, the *râle* is important, and may assist in confirming a diagnosis; but in consequence of its frequent absence, and of the difficulty which must exist in distinguishing it from an ordinary sub-crepitant *râle*, it loses a good deal of the value which would otherwise attach to it.

A knowledge of the morbid changes produced by emphysema, affords a ready explanation of the peculiar character of the respiratory movements and sounds, as well as of the other physical signs and symptoms of the disease. It also enables us to explain the occurrence of the secondary consequences which ensue in the progress of the affection; a subject to be dwelt on hereafter.

The lungs being the seat of general expansion, the thorax is kept abnormally distended: hence, between the point to which it is contracted at the end of expiration, and that of its extreme distension, there is much less difference than in a state of health. The lungs undergo but little enlargement at each inspiratory act. There being no impediment to the passage of the air to the extreme parts of the lungs—the air-sacs—inspiration is rapidly accomplished. Not so, however, the expiratory act. The pulmonary tissue has, in great measure, lost its elasticity, and reacts but slowly after distension. As collapse, or elastic reaction, of the lungs, is one of the most important elements in expiration, any imperfection in it necessarily results in a laboured, slow, and ineffectual expulsion of the air. Further, in advanced states of the disease, when the pulmonary tissue is riddled with perforations, there is an additional cause of impediment to the exit of air from the air-sacs, from the side currents which must necessarily be produced. It is obvious that, under the conditi onsI have described, the respiration can be, at the best, but imperfectly performed; and that there is no room for increased expansion of the chest walls, nor any possibility of the frequency of the respiratory acts being much increased, to meet the requirements of extraordinary exertion. As long as muscular action is slight, and the circulation is quiet, the lungs may perform their work without producing distress: but anything which tends to accelerate the circulation immediately upsets the balance, and gives rise to dyspnœa and imperfect aeration of the blood.

M. Louis has endeavoured to account for the dyspnœa in emphysema on the supposition that the walls of the air-vesicles (air-sacs) are thickened, or hypertrophied, in the disease. It is quite obvious, as he remarks, that a structural alteration of this kind would interfere with the reciprocal action between the air and the blood in respiration, and would render that action less perfect than in a state of health. But, setting aside the fact, which Louis himself admits, that no proof has ever been afforded of the existence of hypertrophy, we can have no difficulty whatever in accounting for the dyspnœa, without the recognition of any such theory. The diminished amount of aerating surface, in consequence of the destruction of the pulmonary tissue, and the imperfect manner in which the air is renewed in the lungs, are quite sufficient to explain the difficulty which occurs in the breathing, either when the circulation is accelerated by exertion of any kind, or whenever an attack of bronchitis occurs.

Not only is there, in emphysema, diminished respiration, but also a diminution in the quantity of blood circulating in the lungs: for the anatomical changes which take place in the pulmonary tissue cause an impediment to the flow of blood through it. It is an ascertained physiological fact, that, when the lungs are in a state of overdistension, the passage of the blood through them is less free than when they are more moderately distended; and a similar result is observed with regard to fluids injected into the lungs, when removed from the body. From this we may gather that, in the earliest stages of emphysema, there is an impediment to the pulmonary circulation; and we find, in fact, that, as the disease progresses, the capillary vessels become ruptured and absorbed, and a considerable diminution takes place in the vascularity of the lung-tissue. Hence we have the pale, anæmic, and dry condition, so characteristic of the emphysematous lung, a condition which serves to explain how it happens that the organ is so rarely attacked with pneumonic inflammation, or even extreme congestion.

An important feature observed in this disease, is, a decrease of the temperature of the body—a condition which results from the diminished quantity of arterial blood circulating in the system, and perhaps, also, from the fact that the blood, which reaches the left side of the heart, has only undergone a partial oxygenation; for both the aerating surface of the lungs, and the power of renewing the air in them, are diminished by the disease. The peculiar aspect of some patients suffering from advanced emphysema, their dull, heavy appearance, the slowness of their movements, and the general torpor of their animal functions, all indicate an imperfect aeration of the blood, and a liability to those secondary diseases which a malperformance of the respiratory function may engender.

CHAPTER XIII.

EMPHYSEMA OF THE LUNGS—THE SEQUELÆ OF EMPHYSEMA, AND THE DISEASES ASSOCIATED WITH IT.

Bronchitis; its General Characters; Sources of Danger in Severe Cases; Deposit of Fibrinous Clots in the Heart, etc. Asthma; its frequent occurrence; its Relations to Emphysema. Disease of the Heart; Parts involved; Causes. Dropsy. General Cachexia and Anæmia. Extravasation of Air into the Areolar Tissue of the Trunk. The Relations of Emphysema with Phthisis, etc.

Amongst the most frequent of the affections associated with emphysema, is, bronchitis. It is rare, indeed, for the former disease to exist for any length of time, without the supervention of the latter. As it is no part of my purpose to consider the subject of bronchitis generally in this chapter, I shall merely allude to the peculiarities it presents when attacking emphysematous patients. In its milder, and more frequent form, it offers no features requiring special comment. Of the treatment appropriate to it I shall speak hereafter. Pathologically, the disease, whether assuming a mild or severe aspect, appears to me one of congestion, or of inflammation of a low type, rather than of an active character. It is attended, when severe, with very profuse secretion; a circumstance, which, coupled with the fact that expectoration is less easily accomplished than when the lung-tissue is in a healthy state, seriously complicates the affection, and increases the danger of death from apnœa. The inflammation sometimes runs on very rapidly to suppuration, and copious purulent expectoration occurs. Even when this has been the case, an examination of the bronchial tubes after death may reveal but little vascularity of the mucous membrane, especially in the smaller divisions, which I have found full of pus, but yet presenting a somewhat pale and anæmic appearance. In the larger bronchial tubes more evidence of inflammatory action will be seen, and especially if the attacks of bronchitis have been frequent.

If these severe cases of bronchitis are not at once appropriately treated, the symptoms very rapidly assume a most urgent character, and life is in imminent danger. Such cases as these, where the early symptoms have been neglected, are occasionally met with, especially in hospital practice, and in the colder seasons of the year. Several have fallen under my notice; and I have preserved notes of some which have been admitted into hospital, and which I have carefully watched till recovery or death took place. Such cases present, for the most part, the following symptoms, when first seen:—The surface of the body is cold, the lips are blue, the whole integument is more or less livid, and the respiration very laboured; there is little or no general expansion of the chest on inspiration, but a rapid elevation of its upper part, with slight protrusion, and in some cases a rapid descent, of the diaphragm; the pulse is quick, weak, and small; the voice is feeble, and sometimes the patient can only speak in a whisper; the chest is very resonant; coarse, moist *râles* are heard all over the lungs; and, if the strength is not too far gone, there is copious expectoration. The mental functions are seriously impaired; in fact, from the gradual blocking up of the bronchial tubes, apnœa is coming on. Unless immediate relief be afforded to such cases, they rapidly go on from bad to worse, and death soon closes the scene. A *post mortem* examination reveals, not only the existence of a large quantity of purulent and muco-purulent fluid in the bronchial tubes, but also the presence of fibrinous clots in the cavities of the heart, and in the large vessels connected therewith.

There are two causes which tend to destroy life in these cases, and it is of the utmost importance that we should bear this fact in mind whenever we have to deal practically with the disease. The first, is, the imperfect aeration of the blood, producing the phenomena of slow apnœa, and resulting from the accumulation of fluid in the bronchial tubes;—the second, is, the constantly increasing obstruction to the circulation of the blood, from the gradual encroachment made on the calibre of the blood-vessels about the base of the heart, and even of the cavities of the heart, in consequence of the formation of fibrinous clots.

The formation, during life, of the fibrinous clots which I have just alluded to, is a very important circumstance in these cases, and affords an explanation of the smallness and feebleness of the pulse, although, at the same time, the heart may be acting vigorously. In some cases, I have seen, after death, a large portion of the cavities of the heart occupied by these deposits, and the calibre of the pulmonary artery and aorta seriously diminished by them. It is obvious that these deposits are only secondary to, and, in fact, produced by, the imperfect aeration of the blood. In proportion as the bronchial tubes become loaded, the pulmonary circulation, and, subsequently, the general circulation, are retarded, and a condition is produced favourable to the coagulation of the fibrine in the blood.

We have no means of ascertaining the presence of these clots during life, except from the general symptoms; these latter, however, often afford us sufficient indications, and I have been able to diagnose, during the progress of a case, the existence of deposits, and to verify the opinion I had formed by a *post mortem* examination.

Asthma, occurring with greater or less severity, is a very frequent attendant on emphysema; so much so, that I am not aware that I have ever seen a case where the organic disease has been of long standing, in which asthmatic symptoms have not supervened. The attacks come on, for the most part, during the night, and, after lasting for varying periods, terminate in the usual manner of such seizures.

An explanation of the occurrence of asthmatic symptoms at night has been sought for, on the supposition that a certain amount of congestion of the lungs takes place during that period, from the position assumed by the body, and the want of activity, during sleep, of the respiratory process. This congestion is supposed to set up an irritation, which gives rise to a reflex spasmodic contraction of the muscular fibres of the bronchial tubes. It is by no means improbable that this view is generally correct; and, especially with regard to emphysematous patients, the occurrence of congestion of the lungs during sleep need excite no surprise: for when we consider the imperfect manner in which the pulmonary circulation is carried on, under the most favourable circumstances, we have no difficulty in understanding how congestions may take place, when the circumstances of position and inactive respiration tend to retard the circulation.

But whatever may be the exact pathological explanation of these attacks, it is certain that they very often occur, and form a most distressing symptom in emphysema. Their frequent repetition leads to that hypertrophied state of the muscular fibres of the bronchial tubes, which

I have described in speaking of the morbid anatomy of the disease.

In all cases of asthma, a careful examination of the lungs should be made, in order to ascertain whether emphysema exist; and I believe that in the majority of cases it will be found. Without entering into a consideration of the general question of the nature of asthma, or of its existence as a purely spasmodic affection, unconnected with any organic disease of the lungs, I may, however, remark, that my own experience leads me to conclude, that emphysema is, by far, the most frequent cause of asthma. Although asthma, commencing as a purely spasmodic affection, may give rise to partial emphysema, I believe that emphysema is more frequently the primary disease. Except the cough, from which asthmatic patients suffer, I cannot see any cause which would be likely to produce emphysema in them. The spasmodic closure of some of the bronchial tubes, and the violent efforts that are made to inspire, do not appear to me probable causes of the dilatation and rupture of the air-sacs; and, for the reasons I have assigned in speaking of the determining causes of emphysema, any theory which regards the disease as a supplementary lesion appears to me erroneous.

It is well known that, in cases of emphysema, secondary affections of the HEART constantly supervene. It has been the opinion of many pathologists that the right cavities alone become affected; but the researches of Gairdner, Lebert, and others have satisfactorily proved that, in the majority of cases, when emphysema is extensive, and of long standing, the cardiac disease is not confined to one side. My own observations tend to confirm this opinion, and to show that the form of heart disease, most frequently associated with emphysema, is a general hypertrophy, with dilatation of all the cavities, especially of the ventricles, for I have never seen a *post-mortem* examination of a case of extensive, and long-standing lobar emphysema, in which the left cavities, as well as the right, were not affected. Lebert, in his *Pathological Anatomy*, speaks of twenty-five autopsies of cases in which there was extensive emphysema; and of these, in sixteen, there was a marked affection of the heart. Of the sixteen, there were nine in which the hypertrophy was general, instead of being confined to the right side; in six there were changes peculiar to general hypertrophy, with mitral disease in addition: and in one there was insufficiency of the aortic valves.

Louis mentions that he met with diseased heart in ten, out of nineteen cases, in which emphysema was more or less present; and Hasse states that, in extensive emphysema, he has only found heart disease absent once.

The frequent occurrence of cardiac disease, in connection with emphysema, is a fact fully established. That the disease is produced as a consequence of emphysema, we can have no difficulty in believing, when we consider the nature of the lung affection, and, especially, that the palpitation, and other cardiac symptoms, are always preceded by distress in the breathing, and other signs, indicative of the pulmonary malady.

Hypertrophy of the ventricles is not the only change which takes place in the heart, for valvular disease is frequently found. The deposits and thickening, which occur about the valves, are no doubt secondary to the changes which take place in the muscular walls, and must be attributed to the general mal-nutrition produced by the pulmonary disease.

When we consider the anatomical arrangements of the pulmonary tissue, and especially of the pulmonary blood-vessels, in emphysema, we can, at once, give a rational and satisfactory explanation of the causes which lead to hypertrophy and dilatation of the right cavities of the heart. The impediment which exists to the circulation through the lungs, in consequence of the physical condition of the lung-tissue, and the imperfect aeration of the blood, together with the diminution in the number of the pulmonary blood-vessels, must necessarily give rise to an overloaded state of the right cardiac cavities, and to increased action on their part.

But the explanation just given will not, I think, fully account for the hypertrophy, so commonly found, of the left ventricle; for there is a diminution in the quantity of blood which finds its way into that cavity, and consequently, rather less call for muscular action than in a state of health. We might therefore infer that we have, in these circumstances, an element of atrophy, rather than of hypertrophy. But morbid anatomy teaches us that the latter usually exists; and it appears to me that we must look, in part, for an explanation of the fact, to the effects produced on the heart by the displacement it undergoes in the disease. This displacement is always the greatest where the emphysema is most extensive, and it is in such cases that the left ventricle becomes most hypertrophied. As the lungs expand, the heart is pushed away from its normal position; and, consequently, the direction of the axis of its cavities is altered with reference to that of the vessels connected therewith. The ventricles of the heart are so placed, in a state of health, with regard to the arteries which issue from them, that no impediment exists to the onward passage of the blood, and the circulation is effected with the smallest possible expenditure of muscular force; but all displacement of the heart necessarily alters the mutual relations between these several parts, and produces an embarrassment of its action—an embarrassment which can only be overcome by more powerful contraction. We consequently find that hypertrophy follows.

This appears to me to be one cause of the hypertrophy of the left ventricle. But another, and perhaps the main cause, exists in the condition of the venous system, and the consequent impediment to the capillary and arterial circulation, which call for increased action of the left heart. The altered position of the heart must also have an influence on the changes which take place on the right side.

In speaking of the symptoms of emphysema, I have alluded to the powerful impulse of the heart, often felt in the epigastric region, as being in marked contrast to the small and feeble pulse, as felt, for instance, at the wrist. A knowledge of the condition of the heart, and of the state of the pulmonary circulation, serves to explain these phenomena. The powerful impulse of the heart is the result of its hypertrophy, and of its embarrassment from position; whilst the small pulse is due to the small quantity of blood which the left ventricle expels at each beat, and its feebleness, to the fact that the force of the left ventricle is expended, in part, in overcoming the resistance which exists to the passage of the blood from the ventricle, in consequence of the altered position of the latter—and in part merely, in distending the arterial tubes. The diminished circulation through the lungs, and the accumulation in the venous system, sufficiently account for the small quantity of blood, which the left ventricle has to react on at each beat.

As a consequence of the disease of the right cavities of the heart, and of the impediment which exists to the flow of blood through the lungs, in emphysema, congestion of the entire venous system results, and is often followed by DROPSY. Cases of emphysema may, indeed, go on for a long time, without the occurrence of any dropsical symptoms whatever;

and in other cases nothing more is produced than a slight œdema of the legs and feet. Where, however, the right cavities of the heart have become much dilated, tricuspid regurgitation ensues, and the dropsy often assumes a severe character.

Emphysema, when it exists in a general form, is attended in its progress with symptoms of CACHEXIA and ANÆMIA. In some cases there is much wasting of the muscular system, even before any dropsical effusions occur. I have seen instances of this kind, especially in young people, where the disease had existed for several years, and where there was a good deal of emaciation and muscular atrophy. This wasting of the muscles and fat sometimes causes a marked contrast between the appearance of the trunk and limbs, and that of the face, which is often somewhat full and puffy—a circumstance resulting, in part, from causes which I have already alluded to, but probably also, in part, from an hypertrophied condition of the respiratory muscles of the face, which are constantly brought into more than ordinary action.

Further, as a consequence of emphysema, we frequently find that the complexion becomes sallow and anæmic, not unlike that which is met with in disease of the kidneys, and other serious organic affections—an important symptom, indicative of an impoverished state of the blood, and general malnutrition.

The occurrence of the secondary consequences to which I have alluded renders emphysema a disease of serious import. I think it has been the practice to look upon the affection in too favourable a light, as regards the consequences which may ensue from it; and to take rather too unfavourable a view, as I shall endeavour to prove hereafter, of the influence which remedial measures may effect for its relief. There can be no doubt amongst those who have carefully studied the disease, that, although in a great many cases it is very chronic, in some it undergoes a more rapid development, and, when extensive, seriously compromises the safety, and tends to shorten the lives, of those who are affected with it. As, however, with organic affections of the heart, patients may, with care, live on for a considerable period, so it is with emphysema; by avoiding those circumstances which may induce acute bronchitis, or which favour the development of the secondary consequences I have spoken of, danger may be averted, and life may be prolonged.

There is another circumstance which I must refer to here, viz., that emphysema is a more serious affection when it occurs in the adult, than in the aged. In the former, the requirements of respiration are much greater than in the latter; the circulation is more active; the quantity of blood in the system is larger, and nutrition is carried on more vigorously. Any interference with so important a function as that of the lungs will necessarily be followed with more serious consequences, in the early and middle periods of life, than in old age. It is especially also in adults, as far as my observations go, that severe cases of bronchitis, such as I have described in a previous part of this chapter, occur, and which so frequently lead to a fatal result.

I have already spoken of the extravasation of air into the areolar tissue of the neck and trunk, in pulmonary emphysema. I have met with only one case in which this accident occurred. The patient was a young man, whom I saw with the late Mr. Swift, of this town, and who had suffered for some time from symptoms of phthisis. A day or two before my visit was made, a puffy swelling appeared about the neck, which soon extended. When I saw the patient, the emphysema had spread upwards, to the face, and downwards, over the chest and abdomen, as far as Poupart's ligament, on either side. Death ensued in a few days; and on making a *post mortem* examination, tubercular deposit was found in the lungs, with only a slight amount of emphysema. The areolar tissue about the root of one of the lungs, and in the mediastinum, was emphysematous, and served to show the source from which the air, infiltrated beneath the skin, had proceeded.

The next subject to which I wish to refer, is, the relations of PHTHISIS to emphysema. I shall not enter into any lengthened consideration of the question, but confine myself, for the most part, to a brief statement of the results of my own observations on the cases that have come before me. An impression has prevailed that emphysema and consumption are incompatible diseases; but I think the pathologists of the present day have abandoned this view, as contrary to the teachings of experience.

That emphysema and tubercular deposit are frequently found in the same lung, has been shown by the observations of numerous writers; and I may adduce the results of my own researches, as tending to confirm the fact. In a large proportion of the *post mortem* examinations of phthisical cases that I have seen, since my attention has been directed to this subject, I have found emphysema present to a greater, or less degree. M. Gallard, who has written an able paper on the question,* states that he also found a similar condition of the lungs in all the cases of phthisis which he examined during the time of his enquiry. The emphysema, he says, does not always exist on the surface of the lung, but is often found in the fissures between the lobes, and even in the interior of the organ. My investigations, with reference to this point, have not been carried out with the same degree of care as those of M. Gallard appear to have been; but I have found patches of emphysema along the borders of the lungs, and on their surface, in the cases to which I have alluded above.

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* "Mémoire sur l'Emphysème Pulmonaire, etc. Archives Générales de Médecine." Paris: 1854.

I have no doubt that these patches of emphysema are produced in the manner I have described, in speaking of the determining causes of the disease, viz., by the act of coughing, usually a prominent symptom in consumption; and that, in the vast majority of cases, the emphysema is a secondary consequence of the tubercular deposit. But an important question arises here, viz., whether tubercle is ever deposited in emphysematous lung-tissue. The fact that the two affections exist in the same lung, by no means answers the question in the affirmative.

Rokitansky, in his *Pathological Anatomy*, remarks, that the venosity and cyanosis, which ensue in emphysema, from the changes that take place in the blood-vessels, etc., constitute the leading grounds for the immunity of emphysematous patients from tuberculosis.

Before this theory, that the condition of the blood in emphysema produces an immunity from consumption, can be admitted, the existence of such immunity must be proved. Although it may be true that, as a rule, tubercle is not often found in patients suffering from general emphysema, yet, that such patients enjoy no complete immunity from the deposit, is shown by the concurrent testimony of numerous investigators. I have seen several instances confirmatory of this view, where, for instance, both lungs were emphysematous—one entirely free from any symptom indicating morbid deposit, but the physical signs and general symptoms pointing to the existence of tubercular disease in the other. I believe that where general lobar emphysema exists, if there be a tubercular diathesis, the so-called venosity of the blood does not prevent the deposit of tubercle. In a patient who died in the Northern Hospital, in June, 1860, I found one lung full of vomicæ; the opposite lung was large, pale, and had all the external appearances of an emphysematous lung. On examination, it was found studded throughout with semi-transparent tubercular matter. A small piece of the lung was carefully examined under the dissecting microscope; and it was at once seen that the tubercular matter had been deposited in the air-sacs and ultimate bronchial tubes. By careful manipulation, portions of the tubercular matter could be drawn from the cavities they occupied, and perfect moulds of these cavities were thus obtained.

But although I cannot agree with the view that the venosity of the blood, in this disease, produces an immunity from tuberculosis, there can be no doubt that the condition of the blood-vessels of the lungs renders emphysematous patients, for the most part, exempt from certain other pulmonary affections, such as œdema, severe hæmorrhage, and pneumonia. It is rare to see œdema of an emphysematous lung; and although a slight amount of blood may occasionally appear in the sputa of patients suffering from emphysema, severe hæmorrhage never occurs. Further, I have in no instance seen an emphysematous lung the seat of pneumonic consolidation. In one instance where emphysema existed, and where death took place from pyæmia, I found a small abscess, with slight inflammation surrounding it, in the substance of one lung; but the remaining portion of the organ was free from inflammatory action.

Inflammation of the pleura by no means unfrequently exists in connection with emphysema, as is proved after death by the presence of adhesions, sometimes of a very firm character. I believe the occurrence of pleurisy, in these cases, must be considered as an accidental circumstance, and that it in nowise results from, or is produced by, the organic disease. In the most extensive cases of emphysema, pleuritic adhesions are not usually found. One result of these adhesions, when extensive, is to prevent the production of emphysema in the parts which lie immediately beneath them.

CHAPTER XIV.

EMPHYSEMA OF THE LUNGS.—TREATMENT OF EMPHYSEMA, AND OF THE DISEASES CONNECTED WITH IT.

Treatment Considered under Two Heads: 1st. Of the Disease itself; 2nd. Of the Secondary Consequences. General principles of Treatment. Remedies to be Used. Dietetic and Regiminal Treatment. Treatment of Bronchitis in its Acute Form. Cases. The Use of Turpentine in Severe Cases. Treatment of Chronic Bronchitis; of Asthma; of Cardiac Disease, Dropsy, etc.

THE general treatment of emphysema requires to be referred to under two different heads. We have, *first*, to take into consideration the treatment of the disease itself, to examine whether the morbid condition of the lung-tissue admits of being checked in its progress, improved, or cured; and, *secondly*, we have to consider the treatment of the various secondary affections which follow as consequences of, or are associated with, the primary disease. And first, with regard to the treatment of the disease itself.

It cannot be doubted, I think, that this question has not sufficiently occupied the attention of physicians, and has not received the consideration it deserves. As I have previously remarked, numerous investigations have been made with reference to the morbid anatomy of the disease, and speculation has been extremely rife as to its determining causes. But in regard to the question of treatment, the end and aim of all pathological enquiry, little appears to have been done; and the affection seems to have been looked upon as one for which but slight remedial aid could be given, except in so far as to relieve the secondary symptoms which follow in its train. It is true that Dr. Stokes and some other writers have expressed their opinion of the possibility of restoring the lung-tissue, in some measure, to a healthy state; but no definite line of practice has been laid down, by which this desirable result could be obtained. In fact, the therapeutical directions in our systematic works have been most meagre; and it is by no means improbable that the pathological views of the disease, entertained by most physicians, have tended to produce this result. The following quotation, from the work of Dr. Stokes, giving his opinion on the question, will probably also express the general view that has prevailed:—"To the practical physician, however, the great point of consideration is, that the disease of the lung is the result of bronchitis; and that for its prevention, alleviation, or cure if that were possible, the treatment must be conducted on this principle." It is true that this was written many years ago, and it is possible that the author of it may have modified his opinion in accordance with our more advanced pathological views; for, as I have already shown, there has been an increasing conviction of the existence of some morbid change in the lung-tissue, as a primary element in the worst forms of emphysema.

On the view we take of the pathology and determining causes of the disease, the whole question of its treatment hinges; for, if we look upon it as simply and solely the result of bronchitic affections, our efforts will be solely directed to the prevention of these attacks; and we shall be apt to consider, when our efforts have been directed to this end, that we have done all that is possible towards the alleviation or cure of the primary disease. This, in my opinion, would be to take a very narrow view of the question, and one neither warranted by clinical investigation nor pathological enquiry.

If, however, we recognize the disease, in its worst forms, as depending on some degeneration of tissue, we may hope to be guided to a more appropriate and successful mode of treatment, than we should adopt by taking a more contracted view of the nature of the affection.

Morbid anatomy affords us a clear insight into the physical condition of the lung-tissue in emphysema: and we may fairly ask ourselves whether we are in possession of any means, by the use of which, we can hope to restore it to a healthy state. That, when the perforations in the walls of the air-sacs have taken place, and the walls themselves have become ruptured and torn, it is possible, by the use of any remedial measures we are now acquainted with, to restore the lung-tissue to a normal condition, we are not warranted in asserting. The perforations, once established, probably always remain; and the lung-tissue, once ruptured and torn, probably rarely undergoes, at the very best, but partial restoration. But whilst these facts are admitted, it is by no means implied that the disease is beyond our control. On the contrary, that condition of the lung which precedes the perforations, viz., the simple distension of the air-sacs, is, I believe, capable of great amelioration; and not only so; but even when the disease has proceeded beyond the stage of distension, although it is less amenable to treatment, a material improvement may in most cases be produced.

If the view I have taken of the pathology of emphysema be correct, viz., that the primary step in the disease is a morbid action, producing a loss of elasticity in the walls of the air-sacs, and, subsequently, their disorganization; and that these changes are the result of a disordered or imperfect nutrition, we may reasonably hope that, by improving the nutrition, the progress of the degeneration may be checked or arrested, and possibly, even, that the elasticity of the lung-tissue may be restored. It is clear that this view involves an important point in therapeutics; and, if correct, will form an equally important basis on which to rest our general principles of treatment.

The main principles, in my opinion, which should regulate our treatment of emphysema—setting aside for the present the management of its secondary affections—are such as guide our practice in all cases in which we have to deal with, what we term, a degeneration of tissue; as, for instance, Bright's disease of the kidneys, fatty degeneration of the heart, etc. We must consider the disease in the light of a constitutional affection, to be treated by constitutional remedies. All measures which tend to invigorate the system, to give tone to the walls of the heart, and to improve the condition of the blood, should be resorted to. In carrying out these indications, we must bear in mind the peculiarities of the malady we have to deal with, and the impossibility of applying, to patients suffering from it, the same matters of detail, in regard to treatment, as we can safely recommend to those who suffer from disease of the heart, or renal affection.

The indications to which I have referred, must be carried out both by medicinal and regiminal treatment; and first, with regard to the former.

Amongst the remedies for internal administration, which I have found the most useful in the disease, is, iron. I was led to employ it from a knowledge of its value in those diseases which are attended with mal-nutrition and cachexia, and especially the two I have alluded to above, viz., Bright's disease of the kidneys, and fatty degeneration of the heart. I have employed it in a large number of cases, of which I have preserved notes, in dispensary, hospital, and private practice; and I have found its administration followed by so much benefit, that I consider it the most valuable medicinal agent we possess for the treatment of emphysema. It is necessary, as is the case in all diseases of a degenerative character, that the use of the remedy should be continued for a considerable time—many weeks, or even months—if the full benefit is to be derived from it. It may be necessary to omit it occasionally for a short time, as symptoms may arise indicating its temporary disagreement with the patient; but no special directions are required on this head.

I am not aware that one preparation of iron is to be preferred to another; whatever form of the drug is found to agree best, should be persevered with. The preparations I have used most are—the ethereal tincture of the acetate of iron; the tincture of the sesquichloride; the sulphate; and the compound steel pill. The first of these forms—the ethereal tincture of the acetate—is a very valuable one, especially when bronchitis is present; as, from its stimulating properties, it acts as an expectorant. I have found it disagree with some patients, who were, however, able to take some other form.

Small doses of quinine, combined with the iron, may be often given with apparently decided benefit. The quinine, no doubt, assists in improving the tone of the digestive organs—a matter of the greatest importance in the management of the disease. Mineral acids may also be given with a similar view. As will be seen further on, I have used iron somewhat largely, in cases of acute bronchitis occurring in emphysematous patients, at that period of the disease when the more urgent symptoms have passed off. Of the mode in which I have administered the remedy under these circumstances, I shall speak hereafter.

I shall not refer, in detail, to the exhibition of remedies which may be called for to relieve any dyspeptic symptoms that may arise. Mild bitters may be sometimes required, and occasionally small doses of a mercurial, with compound rhubarb pill, or some other aperient. Dyspeptic symptoms should, however, rather be met by general management and diet, than by the administration of drugs. Practically, I for the most part confine myself to the medicines I have mentioned above, except in regard to the sparing use of expectorants, and remedies addressed to the bronchitic and asthmatic symptoms, from which the patients generally suffer more or less. The iron must not be given in large doses, nor yet very frequently; but in the same manner, and on the same principles, as we give it, and other tonics, in such diseases as I believe emphysema to be closely allied to. In some cases, where there has been a good deal of wasting, I have found cod-liver oil of service.

Strychnia has been recommended as a remedy for emphysema. It has been given with the view of improving the tone of the muscular fibres of the bronchial tubes. Dr. Walshe states that he has tried it in a small number of cases, both endermically and by the mouth, in sufficient doses to produce obvious effects on the voluntary muscles, without in the slightest degree modifying the symptoms of the disease.

The failure of strychnia is not at all to be wondered at; the remedy must have been given under an entirely erroneous view of the pathology of emphysema. The affection is not one primarily affecting the bronchial tubes, but the lung-tissue itself; and if any secondary consequences ensue, with reference to the bronchial muscles, they are those of spasmodic contraction, rather than of paralysis.

The regulation of the diet in emphysema, and the general management of the disease, are of as much importance as the medicinal treatment. On these points it will be sufficient to indicate a few principles, without entering into any minute details. The diet should be nourishing, and a moderate amount of stimulants should be allowed, depending more on the habits of the patient than on any particular rule. The food should be easy of digestion, and of a dry, solid character—*i. e.*, possessing plenty of nutritious matter, in proportion to its bulk. Overloading the stomach should be especially avoided, as well as everything which has a tendency to produce flatulence; for these conditions always give rise to dyspnœa, and often to palpitation of the heart. When we consider the effects of an overloaded stomach, or of distended bowels, in impeding the descent of the diaphragm, and thus interfering with the action of the lungs, and in embarrassing the movements of the heart—even when both these organs are in a normal state—we need be in nowise surprised that more serious symptoms, and greater distress are produced by the same causes, when the respiratory surface of the lungs is diminished, and the heart's action is already laboured. It is doubly important, in emphysema, that the nature of the food should be carefully looked to; in order that, not only the primary digestion may be well performed, and no local cause may exist to impair the function of the lungs, but that the secondary process of assimilation may also be as perfect as possible, so as to ensure a supply of healthy blood for the nourishment of the lung-tissue. But further; an additional reason exists for the regulation of the diet, in the fact that emphysematous patients often suffer from dyspepsia.

An important principle to be borne in mind, in the treatment of emphysema, is to endeavour to keep the blood moderate in quantity but rich in quality. It is obvious that, when the aerating power of the lungs is diminished, the smaller the quantity of blood sent to them, the more completely will it undergo its proper chemical changes, and the less will be the dyspnœa produced. It is with this further object in view that I have recommended that the food should not be of a bulky or watery character, but very nutritious, and as digestible as possible. In accordance with the same principle, it is desirable that the quantity of liquids taken should be small, in order that the blood-vessels may not be, even temporarily, overloaded with a watery fluid. As a drink, wine, or wine and water, is to be preferred to malt liquors, which have a great tendency to produce flatulence, especially when the digestive organs are weak. Another principle of treatment to be looked to, is that of giving the lungs as little work as possible, and letting the patient breathe a pure air: so that the greatest amount of aeration of the blood may be produced, in proportion to the quantity of air inspired. Unfortunately, in the treatment of visceral disease, the physician cannot give the organ he has to deal with absolute rest from the performance of its function. Unlike external parts, which may be placed almost in a state of physiological rest, the internal organs, the lungs, the heart, the liver, etc., and more especially the two first, can no know period, however short, of absolute repose. But although this result cannot be fully accomplished, it must nevertheless be aimed at in many visceral diseases, and more especially in emphysema. All violent exercise, or great physical exertion of any kind, must be strictly prohibited. It is not that all exercise is to be proscribed; on the contrary, a moderate amount is to be recommended, and this should be increased as we find the condition of the lungs improving. Exercise in the open air, moderate walking, carriage exercise, etc., form important adjuvants in the general treatment of emphysema.

In a very large proportion of the cases of emphysema which come under our notice for the first time, the symptoms of bronchitis, either acute, sub-acute, or chronic, are present. Most frequently the bronchial inflammation is chronic; but occasionally it assumes symptoms of a very severe character, such as I have described in a previous chapter.

In the treatment of all forms of bronchitis occurring in emphysema, I believe the greatest caution should be used in the administration of remedies which have at all a depressing character: and my experience does not lead me to coincide with an opinion expressed in some works on diseases of the chest, as to the great value of local bleeding, blistering, and tartar emetic, in the affections we are now considering; nor do I consider them as essential agents in their removal. I have never found it necessary to abstract blood in any way; and although blistering, or some form of counter-irritation, is often of the greatest service, I believe that few cases require, or are benefited by, the exhibition of tartar emetic. It is very possible that, in the earliest periods of an acute attack, when the bronchial mucous membrane is dry and swollen, a few doses of antimony may be given with advantage: but the period when this remedy is likely to be of service is soon gone by; and when once secretion, always profuse in these cases, has occurred, my belief is that tartar emetic is a remedy which ought not to be administered. Further, in the treatment of the less acute forms of the affection, I have never found it necessary to administer antimony; and my opinion is, that all cases will be most satisfactorily managed without it; and that, even in the use of ipecacuanha, we should be cautious and sparing.

I do not wish to enter, here, into a discussion of the general principles on which bronchitis should be treated; but simply to refer to the indications which should guide us in those cases, in which, it occurs in connection with emphysema; my observations must, therefore, be considered as entirely referring to such cases. We must never forget that our patients are suffering from a depressing organic disease; and that they labour under a physical difficulty to get rid of the secretion which accumulates in the bronchial tubes. We must bear in mind that we have to guard against, and obviate, the tendency to death, first, from slow apnœa, and, secondly, from the deposit of fibrinous clots in the heart and great blood-vessels.

I have spoken of the dry condition of the bronchial tubes in the early stages of bronchitis, and the possibility that antimony may be usefully administered at that period; but the fact is, that, in consequence of the more or less chronic inflammation, which is almost invariably present in emphysematous patients, the bronchitic malady creeps insidiously on from the chronic, to the sub-acute, or acute form; and it usually happens that, when we are called to these cases, we find the bronchial tubes more or less loaded with fluid.

What, then, is the general line of practice which ought to be adopted, in these bronchial affections; I believe it should be essentially of a stimulating and sustaining character, and that all measures which have a tendency to depress the patient, or to diminish his expectorating power, should be avoided: whilst, on the other hand, those should be resorted to which tend to obviate death from apnœa.

These indications will be most effectually carried out by the use of such medicines as ammonia, ether, squills, and in some instances ipecacuanha, etc.; and by the free, persistent, and regular administration of alcoholic stimulants—wine or brandy. There is no better mode of exhibiting the medicines I have mentioned, especially in the earlier stages of the disease, or when thirst or irritability of the stomach is present, than in combination with an effervescing saline. They may also be given, in a later stage, with decoction of senega, or any other preparation of a similar character.

The alcoholic stimulant must be given at regular intervals — every hour, or every two, three, or four hours, according to the urgency of the symptoms; and we must not be deterred from administering it, although the skin be hot and the pulse rapid: in fact, as a general rule, the more rapid the pulse, the more freely stimulants should be given. In many cases, I believe, wine is to be preferred to brandy, or any other form of alcohol. In other cases, however, from the urgency of the symptoms threatening apnœa, it is necessary to give the more powerful stimulant; and it may happen that wine is not well borne by the stomach—in which case also brandy must be used. In one of the cases detailed below, this latter circumstance occurred; the patient rejected everything that was swallowed, until a few doses of brandy were given, which checked the vomiting at once, and after being continued for two days, wine and food were again borne.

In these acute cases, as soon as the symptoms have begun to improve, and whilst the secretion of the bronchial tubes is still profuse, I have seen great benefit follow the administration of iron, in frequently repeated doses; as, for instance, ten, fifteen, or twenty minims of the tincture of the acetate, every four or six hours. Under this treatment I have seen the quantity of mucus expectorated rapidly diminish, and all the other symptoms improve. In sub-acute and chronic cases, we may begin very early with iron; but, as I shall have to speak of it hereafter in connection with such cases, I shall defer what I have to say of its action.

In addition to the measures which I have recommended, counter-irritation should be resorted to. Blisters are often very beneficial, but in many cases I think some milder form of application, more extensively used, is to be preferred; as, for instance, croton oil liniment, or some other stimulating embrocation, freely rubbed in all over the front of the chest. Turpentine fomentations are also of great service, especially in the early periods of the attack.

The diet should be carefully attended to; it should be of a digestible and nutritious character, and regularly administered. The bowels should be kept open, for all accumulation in them has a tendency to distress the breathing, and embarrass the action of the heart. A purgative, mercurial or otherwise, may be given in the first instance; but, for the most part, enemata should be used, in preference to aperients given by the mouth.

As illustrating the principles which I have endeavoured to lay down in the preceding remarks, for the treatment of acute bronchitis occurring in emphysematous patients, I shall quote the following cases, which I have selected from my hospital note-book, as being well-marked instances of their kind.

Both the patients referred to had, at one time, symptoms of so severe a character, that death appeared imminent; both were treated with similar remedies, and both made a satisfactory recovery. They were carefully watched by the students, the house-surgeons, and myself, and notes of their progress were taken from day to day. Although these notes are somewhat lengthy, I prefer to give them without alteration, and with all the details which were drawn up at the time.

Mary J., aged 20, single, was admitted into the Liverpool Northern Hospital on April 22nd, 1862. She said she had suffered for some time, but only to a slight extent, from cough and shortness of breath; she had never been laid up till about a week before admission, when she was obliged to take to her bed, and had been attended by a surgeon from a dispensary.

On admission, the countenance was dusky; the eyes suffused; there was great general distress, with urgent dyspnœa. The pulse was 148, and very feeble; the respiration was rapid. There was rather increased resonance all over both lungs, with resonance in the cardiac region. The impulse of the heart could not be felt. There was scarcely any lateral expansion of the chest on inspiration. The thorax was raised by the cervical muscles at each inspiration, and the lower part of the abdomen forcibly protruded. Coarse and subcrepitant râles were heard all over both lungs, with prolongation of the expiratory murmur. The tongue was furred; the skin hot; and there was great thirst. She was ordered an ounce of port wine every four hours, and an ounce of the following mixture every four hours:—Carbonate of ammonia, ʒ ss; spirit of chloric ether, ʒ ij; water, ℥ viij. For diet, strong beef-tea was ordered.

On the 23rd, she had had no sleep. The bowels had acted freely. The pulse was 144, the respirations were 48 per minute. She had expectorated a large quantity of frothy mucus. She was ordered to take an ounce of port wine every three hours, and to continue the mixture.

On the 24th, the pulse was 136; the respirations were 44. She had been purged several times. The chest symptoms were somewhat relieved. The mixture was stopped on account of the purging, and the wine increased to an ounce and a half every three hours.

On the 25th, she had slept well. The bowels were still rather relaxed, and the skin was somewhat hot. The tongue was cleaner, and she said she felt better. The pulse was 148; the respirations were 48. There were still moist râles all over both lungs. She was ordered to continue the wine every three hours, and to take an ounce of the following mixture every four hours:—Solution of acetate of ammonia,

℥ iss; tincture of opium, ʒ ss; water, to ℥ vi. Croton oil liniment was ordered to be rubbed over the chest, and fifteen minims of Battley's solution to be given at night.

On the 26th, the pulse was 120; the respirations had fallen to 32. She had passed a fair night; the bowels had acted once; there was less expectoration. In the evening she complained of feeling very sick; the administration of some brandy relieved her. She was ordered to continue the wine and mixture, and to have twenty minims of solution of morphia at bedtime.

On the 27th, the pulse was 120; the respirations were 40. She had slept well; the bowels were quiet; and the tongue clean. The sputa were frothy, and less abundant. The mixture was stopped. She was ordered to continue the wine, to have another application of the croton oil liniment, to take twenty minims of laudanum at bedtime, and to have some meat.

On the 28th, the pulse was 120; the chest-symptoms were much relieved, but the expectoration was still copious. She was ordered to take ten minims of the ethereal tincture of the acetate of iron every four hours, and to continue the wine.

On the 29th, the pulse was 118; the breathing was better; she had slept well, and had taken her food.

On May 1st, she was improving; coarse crepitation was still heard at the back of both lungs.

On the 2nd, the pulse was 100; the respirations were 32. The dose of tincture of iron was increased to fifteen minims every four hours, and some laudanum and squills were added to it.

On the 3rd, the pulse was 106, and feeble. There was less sputum, and it was more tenacious. The wine was reduced to eight ounces daily.

On the 6th, she was up. The pulse was 100, sitting.

The tongue was clean, and the cough gone. There was very little expectoration. The appetite was good. She was ordered to take the iron mixture three times a day, and the wine was reduced to six ounces daily. From this date she improved rapidly.

On the 10th, the pulse was 68; the breath-sounds were free from râles, but there was a prolongation of the expiratory murmur, especially over the back of the lungs.

On the 15th, she was discharged.

Mary B., aged 19, was admitted into the Liverpool Northern Hospital on April 21st, 1862. She said she had suffered for five or six years from cough and difficulty of breathing, but had never been laid up. She was as well as usual on April 17th, four days before admission. On the following day, she had a headache, but did not take to her bed till the 20th. No treatment had been adopted.

On admission, the skin was hot and perspiring; the pulse was 128, and small. There was urgent dyspnœa. The lips were somewhat blue, and the eyes suffused. The breathing was superior thoracic, with but little or no action of the diaphragm.* The upper part of the chest was very prominent; and there was increased resonance over both lungs, with resonance of the cardiac region. Coarse and subcrepitant râles were heard all over the chest, with prolonged expiratory murmur.

She was ordered an ounce of port wine every four hours, and a mixture containing carbonate of ammonia and chloric ether; with strong beef-tea.

On the 22nd, she had passed a restless night, and had vomited both her food and wine. The pulse was 114; the respirations were 48. There was copious muco-purulent expectoration. The general symptoms were about the same as on the previous day. She was ordered to omit the wine and ammonia, and to take a teaspoonful of brandy every half hour, till the sickness was stopped.

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* The movements of the chest, in the two cases I have quoted, serve to illustrate the different characters of the respiration referred to in the chapter on Physical Signs and Symptoms.

On the 23rd, she said she had not vomited since she began to take the brandy, of which she took four or five doses. She had rested better. The countenance was improved; the pulse was 118; respirations 48; the expectoration was still copious. The urine was loaded with lithates, and contained a small quantity of albumen. She was ordered half an ounce of brandy every three hours; and four ounces of port wine during the day.

On the 24th, she was better, took her food, and had not been sick.

On the 25th, she had passed a good night; the sputum was still abundant, but more frothy; the pulse was 124; the respirations were 48. She was ordered eight ounces of port wine daily, and an effervescing saline with carbonate of ammonia.

On the 26th, the urine was examined, and found free from albumen.

On the 27th, croton oil liniment was ordered for the chest.

On the 28th, she was much improved. The pulse was 100; respirations 40. She had taken her wine and ammonia regularly. There was but little dyspnœa; and the râles over the lungs were coarser. The expectoration was still copious. She was ordered to omit the ammonia, but to continue the wine, and to take ten minims of the ethereal tincture of the acetate of iron every four hours.

On May 1st, the sputum was less copious and more tenacious. The pulse was 100; the respirations were 32; and the appetite was good.

On the 2nd, the dose of tincture of iron was increased to fifteen minims every four hours.

On the 3rd, the pulse was 90; the respirations were 25; the tongue was clean, and the expectoration much diminished. She complained of the cough giving her some trouble. She was ordered twenty minims of tincture of iron and ten minims of laudanum three times a day, and meat diet. The wine was reduced to six ounces daily.

She recovered rapidly from this date, but was kept in the house for some time to be treated for the emphysema. She took a course of iron, with good diet; and under this treatment she gained flesh, and her cough and dyspnœa materially improved.

She left the hospital on June 9th.

The cases I have just detailed serve to show the beneficial effects which may be produced, on certain severe forms of bronchitis, by the free and persistent use of alcoholic and other stimulants. I do not wish, in this chapter, to enter into any general observations on the therapeutic value, or mode action of alcohol; and I shall therefore content myself with remarking that, in watching the results of the remedy in cases similar to those I have described, and, in fact, in all so-called asthenic inflammations, there is nothing more striking than the effects it produces on the circulation; for we find the pulse gradually diminishing in frequency and increasing in power—a sure sign that the patient is benefiting from the treatment adopted, and that the disease is taking a favourable course. In the management of all inflammatory disease, the character of the pulse constitutes one of the most important guides as to the propriety of administering stimulants, or of withholding them; and it cannot, I think, be doubted that, if our knowledge of the indications afforded by the pulse were more accurate and defined, our treatment of these acute affections would be characterized by more uniformity and a greater measure of success.

It occasionally happens that these cases of bronchitis, either from neglect or extreme severity, assume a character which I have described in a former chapter, when the surface of the body becomes cold, and the pulse exceedingly small and feeble; and when, from the accumulation of fluid in the bronchial tubes, and the inability of the patient to expectorate, apnœa is threatened. My experience of the treatment of cases which have reached this degree of severity, is, that the ordinary stimulants, alcohol, ether, ammonia, etc., have but little influence in rallying the patients from their semi-asphyxiated condition. I have given brandy, and other stimulants, in moderate, and in large doses, at frequent intervals, and, apparently, without the least benefit being produced. On making a *post-mortem* examination of cases of this kind, I have found, as I have previously stated, not only that the bronchial tubes were loaded with fluid, but that the cavities of the heart, and of the great blood-vessels connected therewith, were more or less occupied with fibrinous clots. In all probability, when cases assume the severity I have described, a large per-centage of them will prove fatal, and we can scarcely expect that medical art can effect much for their relief. There is, however, one mode of treatment which I have found successful, when alcoholic and other stimulants have failed, to which I think it worth while to allude.

The practice I refer to consists in the administration of moderately large doses of turpentine, on a plan suggested, I believe, by Dr. Corrigan, of Dublin. This medicine has often been recommended in diseases affecting mucous membranes, and in some cases of hæmorrhage its value is unquestionable; but I am not aware that it has been much used in such cases as I am referring to.

I will briefly relate a case in which this treatment was adopted, and in which it produced the most satisfactory results.

Peter C ——, 22 years of age, a sailor, was admitted into the Liverpool Northern Hospital on the 22nd December, 1860, with acute general bronchitis and emphysema. When first seen by me, about noon on the day of admission, he was suffering from all the symptoms I have elsewhere described, and appeared to be rapidly sinking into a condition of apnœa. Alcoholic and ethereal stimulants had been already given, and were freely continued for several hours. Towards the evening, about four or five o'clock, I saw him again. The symptoms had not at all improved, but were rather worse than better. He was ordered dram doses of turpentine in aromatic water every two hours. The symptoms began immediately to improve, the dyspnœa became less, and the skin warmer and of a more healthy hue. The following day, as the turpentine had produced a little strangury, it was omitted. The patient at once began to relapse, and the symptoms soon became as threatening as ever. A half-ounce dose of turpentine was ordered. Marked relief to the lung symptoms followed its administration; but as soon as its effects had passed off, viz., in about four or five hours, a relapse ensued, and another half-ounce dose of turpentine was given, and followed by others. In this way five or six half-ounce doses of the medicine were taken. The result of each dose was, that great relief was given for a time; but, as the effects of the turpentine passed off, the bad symptoms returned, to be again relieved by a further dose. After each dose, the patient appeared stronger, and the interval before the subsequent one was given was prolonged. At the end of five or six days from his admission, no further relapse took place, and the turpentine was stopped. During the time he took the turpentine, he was also taking alcoholic stimulants; these, no doubt, acted beneficially, but the oil seemed to be the great remedial agent.

This case was very carefully looked after by the house surgeon, who administered the turpentine, under my directions, according as it was required; and I could not help thinking at the time, and I am still of opinion, that the man owed his life to the assiduity with which he was watched, and the repeated exhibition, at the right moment, of the remedy which was found to act so powerfully on him. He suffered rather severely from strangury whilst he was taking the oil; but this symptom subsided as soon as the medicine was discontinued.

The following were the results which followed the administration of the turpentine, and which were apparently produced by it. The expectorating power of the patient was increased, the dyspnœa was relieved, and the effusion into the bronchial tubes was diminished. After the turpentine was left off, and the urgency of the symptoms had subsided, ethereal tincture of the acetate of iron was given, and continued for some weeks. Under this treatment a steady improvement took place, and the patient was discharged six weeks after his admission, his general appearance being then much improved, his dyspnœa lessened, and his power of taking exercise far greater than it had been, he said, for a long time.

It will be observed that, in quoting the above case, I have not spoken positively as to the number of doses of turpentine the patient took, nor as to the number of days he was under this treatment. I have mentioned five or six. The fact is that the early notes of the case are lost; and although all the statements are in the main correct, yet, in the above matters of detail, I have been unable to speak in that positive manner which is always desirable when cases are quoted. This does not, however, affect the argument as to the value of the remedy made use of.

I cannot strengthen the case by adducing others to support it; I give it, therefore, as an isolated one, for simply what it is worth. The results of the treatment were so satisfactory, that I am of opinion that, in these extreme cases, when other measures fail, we may fairly have recourse to it. Caution is, however, necessary, in the use of the remedy; it must not be given in large doses at first, or it may produce great depression. The best plan is to begin with half dram, or dram, doses, every two or three hours; and then, if necessary, to give the larger dose less frequently.

In a case very similar in symptoms to the one just quoted, a half-ounce dose of the oil was given at first; it produced some sickness, but had no effect in rallying the patient from his semi-asphyxiated condition, and he subsequently sank. After death, both lungs were found highly emphysematous throughout, and firm fibrinous clots existed in the cavities of the heart, as well as in the pulmonary artery and aorta; a condition probably precluding all chances of recovery.

The administration of an ipecacuan emetic in bronchitis, for the purpose of relieving the overloaded bronchial tubes, is sometimes productive of great benefit, and might be of service in those cases of threatened apnœa which I have alluded to. Not having had any practical experience of it under these circumstances, I cannot speak as to its value.

I will now, briefly, refer to the treatment of the chronic form of bronchitis, which is so constantly associated with emphysema; and as we very frequently find asthmatic symptoms co-existent with the bronchial inflammation, and are often called upon to treat the two affections simultaneously, it will be well to consider the measures which are appropriate for their relief at the same time. As, however, I do not wish to enter into the general question of the treatment of asthma, my remarks on this head will be very brief.

With regard to the bronchitic symptoms, much good can be effected by counter-irritants and expectorants, such as squills, etc. Croton oil liniment, or some other embrocation, should be rubbed over the chest from time to time, as circumstances may require, and it will be rarely found that relief does not soon follow.

In the treatment of these cases, we must bear in mind that, in almost all emphysematous patients, there is, at all times, more or less congestion or inflammation of the bronchial mucous membrane, and that this is always attended with increased secretion. Our object must be, not simply to adopt such treatment as will tend to relieve a present inflammation, and enable the patient to get rid of the excess of mucus which troubles him; but we must further endeavour to act on the relaxed mucous membrane, and give tone to its capillary vessels, so as to obviate the condition which causes these constantly recurring attacks. Although, primarily, expectorants are called for, yet it is desirable to disturb the stomach as little as possible with nauseating drugs, especially if the digestive organs are weak. The regimen and treatment should be of a decidedly tonic character, and carried out on the principles which I have recommended in speaking of the general treatment of emphysema.

I have already referred to cases of bronchitis in which I have administered iron, when the more acute symptoms had passed off, and I have spoken of its value in emphysema itself. Independently of the influence which the remedy seems to have on the pulmonary tissue, I believe it produces a most beneficial effect on the congested and relaxed bronchial tubes in sub-acute and chronic bronchitis. In the cases above referred to, it was given somewhat largely when the more acute symptoms had subsided. In other cases, of a sub-acute, or chronic form, I have been in the habit of giving it, in doses of from ten, to fifteen minims, of the ethereal tincture, or the tincture of the sesquichloride, three times a-day; and, from having very frequently administered it, and seen the rapid manner in which profuse secretion from the bronchial tubes has been checked, and the other symptoms relieved, it has appeared to me to be the best medicine we can give in such cases, with the view of bringing about the secondary result which I have adverted to above. It may be combined with expectorants, or given with wine, or with a bitter tonic, when these are indicated. Besides its general effect, it appears to act as a local astringent on the bronchial mucous membrane, and to possess, in suitable cases, advantages over all other remedies of a similar character with which we are acquainted.

For the relief of asthmatic symptoms occurring in emphysema, I have generally found most benefit from the administration of the ethers, ethereal tincture of lobelia, and especially from the smoking of stramonium and tobacco. I generally recommend patients to smoke stramonium just before going to bed; and I have very constantly found that this has had the effect of giving them a good night, and of preventing them from being disturbed by an attack of dyspnœa. Tobacco I have found very useful in some cases, especially with patients who were not previously in the habit of smoking it; but, as far as my experience goes, its influence over the affection is not as lasting as that of stramonium.

If the measures I have referred to should fail to produce relief, other remedies which have been at various times found useful in asthma may be resorted to.

It is no part of my purpose to consider any special treatment which may be required for the cardiac disease, dropsy, etc., which follow as secondary consequences of emphysema. If the general principles I have referred to for the treatment of the primary affection be carried out, I believe the best means will be adopted to prevent both that state of the heart which is followed by dropsical effusions, and that condition of the system at large which gives rise to anæmia and atrophy.

The preceding chapters on Emphysema were published in a separate volume six years ago. The opportunities, which I have since had, of further observation and enquiry, have tended to confirm the opinions I have expressed of the nature and treatment of the disease.

CHAPTER XV.

ŒDEMA OF THE LUNGS—PULMONARY APOPLEXY.

(CLINICAL LECTURE.)

GENTLEMEN,—There are two pathological conditions of the lungs to which I wish to refer in the present lecture, viz., *œdema of the lungs*, and, so-called, *pulmonary apoplexy*.

First, of *œdema of the lungs*. It is, more especially, to its anatomical characters, than to any other point in connection with it, that I wish to direct your attention.

It is a diseased condition of, by no means, infrequent occurrence, and one of considerable importance. It occasionally becomes the proximate cause of death, when associated with general dropsy.

It is characterized by an effusion of watery, and, sometimes, slightly sanguineous fluid into the substance of the lungs. But I must explain to you what is the exact seat of the effusion. You will find that pathologists, for the most part, describe the affection as consisting in a serous infiltration of the parenchyma of the lungs, and its interstitial tissue, from which you might infer that the fluid infiltrates the areolar tissue throughout the lungs, just as the fluid of anasarca infiltrates the sub-cutaneous areolar tissue of the body generally. But this is not the case. There is, as I have already told you, no such thing as areolar tissue between the air-sacs of the lungs; and the view, which I have just mentioned, of the morbid anatomy of pulmonary œdema has been entertained under a false notion of the anatomy of the lungs. The seat of the œdema is the air-sacs of the lungs; it is into them that the fluid is poured from the pulmonary vessels. No doubt the walls of the air-sacs, which contain those vessels, are swollen, more or less, by the presence of fluid, but inasmuch as they contain no areolar tissue, and consist of a homogeneous membrane, enclosing elastic fibres, there can be no such infiltration as some authors have described—no infiltration similar to that of ordinary anasarca. Wherever there is areolar tissue in the lungs, as, for instance, around the bronchial tubes, the pulmonary vessels, and the lobules, effusion will be found; but, I repeat, that the essential seat of pulmonary œdema is the same as that of pneumonic exudation—the air-sacs. When œdema exists to a large extent, and the air-sacs of a considerable portion of the lungs contain this watery effusion, you can easily understand how the aeration of the blood is interfered with, and dyspnœa is produced.

Œdema of the lungs, whenever it is present to any extent, is not a primary disease. It is secondary to some other pathological condition, and has a passive or mechanical origin. It is for the most part associated with general dropsy, whether depending on some morbid state of the blood, as in Bright's disease, or on some affection of the heart. Further, it occurs in emphysema of the lungs where there is a dilated and weakened condition of the right cavities of the heart. It often becomes, in cases of this kind, the proximate cause of death—the effused fluid by degrees fills the air-sacs and finer bronchial tubes, and thus interferes with the process of respiration, and ultimately produces death by slow apnœa.

Œdema of the lung is frequently associated with effusion into the pleura—hydro-thorax—both morbid conditions being part of a general dropsy; you must not, however, confound the two conditions with each other.

The lower parts of the lungs are, as you might naturally expect, the most frequent seats of œdema, but is is occasionally met with throughout the whole of the lungs.

We have recently had in the hospital, some very marked instances of the affection which I drew your attention to, at the time the patients were here. One was Kenneth M'K——, 60 years of age, in K ward. This man was suffering, when admitted, from general anasarca, and the symptoms of emphysema of the lungs. Moreover, there was a systolic murmur at the base of the heart. On examining the chest, we found some dulness behind, and a crepitating râle. This latter auscultatory sign is a very important one. I have already referred to it in speaking of the crepitation of pneumonia. In the case of another patient, John S., in the same ward, the second instance I wish to refer to, the crepitation was well marked. This man, you will recollect, was admitted on the 8th of February last, and was suffering from symptoms of emphysema of the lungs and general dropsy, without any valvular disease of the heart, or any affection of the kidneys. He was expectorating a large quantity of frothy, watery fluid, and on examining his chest, we found distinct crepitation, especially over the base of the right lung. The crepitation did not, as far as my ear could detect, differ in anywise from that which is met with in pneumonia, and, as it persisted for some time, we had ample opportunities of listening to it. It had the peculiar crackle characteristic of pneumonic crepitation, and it was heard during inspiration only. But although I pointed this out to you at the time, you may recollect I said, that I had no doubt as to the cause of the râle, that, in fact, it was due, not to the presence of pneumonia, but of œdema. The dropsical effusions which we found in other parts of the body cleared up all doubt on the point.

There is another patient, now in the house, John V., in L ward, suffering from hypertrophy, and valvular disease of the heart, in whom there is distinct crepitation heard at the base of both lungs, the result, I have not the slightest doubt, of œdema. When the patient was admitted he had anasarca of the lower limbs, but the rest and treatment, which he has had since he came in, have caused this to disappear. There has been no marked dulness at the back of the lungs, and the area over which the crepitation has been heard has gradually diminished, so that there can be little doubt that the œdema of the lungs is much less than it was. The crepitation in this case was of a fine character, heard only during inspiration, and, by the ear, could not be distinguished from that produced by pneumonia. But the general symptoms pointed to its true nature.

The symptoms and physical signs of œdema of the lungs are by no means of a distinctive character, but still, by exercising proper care, you will rarely have much difficulty in recognizing the disease. Dyspnœa, to a greater or less extent, is always present; there is a short cough, with a watery expectoration; and a sense of oppression, or of weight in the chest, is generally complained of. Moreover, there is an absence of the natural resonance on percussion, and on auscultation, you hear a rhonchus more or less closely resembling the crepitating râle of pneumonia, and, in some cases, not to be distinguished from it. These symptoms are usually associated with general dropsy, the existence of which would prevent you from making an error of diagnosis.

In the treatment of the affection, you must be guided by the nature of the original disease to which the œdema is secondary— diuretics, or tonics, or both combined, are the main agents to be relied on, your choice of them depending on the general condition of your patients and their primary malady.

And now, with regard to *pulmonary apoplexy*. The term is, I think, a very unfortunate one; it means, in fact, pulmonary hæmorrhage, with the blood retained in the lungs. Two kinds of pulmonary apoplexy have been described —one, in which the blood is poured into the air-cells; the other, in which it is extravasated into the areolar tissue of the lungs. Now, the second form I have referred to has no real existence, except in so far as regards the areolar tissue of the bronchial tubes and blood-vessels. In true pulmonary apoplexy the blood is poured into the air-sacs, and, possibly, destruction of their walls sometimes ensues.

If you take a piece of lung, which is the seat of pulmonary apoplexy, and make an incision through it, and then examine it with a strong lens, you will find that it has a somewhat granular appearance, due to the fact that the blood has coagulated in the air-sacs. If you now either dip the piece of lung into rectified spirit, or pour a little spirit over the cut surface, you will be able to make out, more distinctly, that it is the air-sacs alone that are distended with blood; and that the walls which surround them are not infiltrated by it. But the best way of satisfying yourselves with reference to this latter point, is, to examine a piece of apoplectic lung, which has been soaked in spirit for some hours, so as to become quite hard. If you place such a piece of lung under a dissecting microscope, you will have no difficulty in tracing out the finest bronchial tubes, and in laying open the air-sacs. You will find that the walls of these sacs have the ordinary appearances — except that they are more blood-stained — which they present in a piece of healthy lung. Further, you will be able to dissect, out of the sacs, the blood which has been poured into them, and which, by the action of the spirit, has become hardened.

In examining the bronchial tubes in a piece of apoplectic lung, I have found them, even to their termination, free from blood; a circumstance which, I think, goes far to prove that any exclusive theory of the production of pulmonary apoplexy from bronchial hæmorrhage, must be considered erroneous. At the same time, I must say that I have sometimes found a small quantity of blood in some of the bronchial tubes.

The extravasated blood is, as I have said, confined to the air-sacs, and, in some instances, to the finer bronchial tubes; it does not make its way into the areolar tissue between the lobules of the lung. The line of demarcation, between an apoplectic lobule and an adjoining healthy one, is quite abrupt; the sheath surrounding the lobule does not become ruptured, nor does the areolar tissue between it and the adjoining sheath become infiltrated.

Such being the seat of pulmonary apoplexy, the question arises as to the vessels from which the blood is derived. Although I do not deny that the bronchial arteries may sometimes be the source of the hæmorrhage, I yet believe that, in the vast majority of cases, it is the pulmonary vessels from which the blood is poured out; the branches of the pulmonary plexus, either from disease, or from an overloaded state, give way, and thus allow of the escape of their contents into the air-sacs. It is quite possible that some cases of pulmonary apoplexy may be produced by a rupture of the vessels of the bronchial mucous membrane, and that the blood may be driven backwards into the air-sacs, so as to distend them; but this is, I think, an exceptional cause of the disease.

Pulmonary apoplexy is, for the most part, associated with cardiac disease, and sometimes it is, and at other times it is not, attended with hæmoptysis. There are no features by which you can certainly diagnose it. If the hæmorrhage be deeply seated, it may give rise to no physical signs which you can recognize; but, on the other hand, if the apoplectic clots be numerous, or near the surface, you may detect a certain amount of dulness on percussion, and a weakness of the breath-sounds, over the accumulated blood, with loud breathing in the neighbouring parts of the lung.

The following case, in which, from the general symptoms, I suspected the existence of pulmonary apoplexy, will serve as a good illustration of the ordinary features which characterize the disease. It is, like œdema of the lungs, quite a secondary affection. How far, however, it may depend on a diseased condition of the capillaries of the lungs, I am not able to say. It is quite possible that there may be an atheromatous state of these vessels in some cases of pulmonary apoplexy. I have examined some of the smaller branches of the pulmonary arteries, in the disease, but I have not detected atheroma in them. The subject is one, however, which requires further investigation.

John R——, aged 49 years, a labourer, was admitted into the hospital on the 11th July, 1867. I did not see him till the 16th, as I was from home at the time of his admission. I found him suffering from great dyspnœa, with dropsical effusion in the legs. His face was puffy, and his lips somewhat blue. There was greatly increased dulness in the cardiac region, and a double murmur was heard at the base of the heart, and towards the right clavicle. The systolic murmur was not loud, but the diastolic murmur was both loud and prolonged. The pulse was jerking. There was distinct crepitation at the back of the left lung. The urine was 1·028, and contained a small quantity of albumen. Before I saw him, he had been treated with purgatives (compound jalap powder), and he was taking 6 oz. of wine daily. I ordered him some decoction of broom, and sweet spirits of nitre. On the 18th July, the dyspnœa and the other symptoms continued. The sputa were somewhat of a pneumonic character. There was coarse crepitation at the back of the left lung, and very fine crepitation at the back of the right, with dulness, and slight bronchophony, at the base of both. The pulse was 104. We could not detect any fluid in the abdomen. I increased the quantity of stimulants, and added some carbonate of ammonia to the mixture, but no improvement followed. On the 19th, the sputa were bloody, and some of them quite pneumonic in character. There was great dyspnœa, with troublesome cough, and the patient had had no sleep. On the 20th, coarse crepitation was heard at the back of both lungs, and the sputa were watery, somewhat adhesive, and contained blood. Finding the patient worse, I ordered him an additional quantity of stimulants. I gave him a tablespoonful of brandy every two hours, and he was dry-cupped between the shoulders. But he did not improve. On the 21st, the dyspnœa was so great that he was obliged to be constantly propped up. The sputa continued watery, frothy, and bloody. He gradually grew worse, and died on the 23rd. During the progress of the case, I expressed an opinion that it was one of pulmonary apoplexy. The presence of valvular disease of the heart, the character of the dyspnœa and of the sputa, and the dropsical accumulations, all pointed to the probability of the existence of pulmonary apoplexy. At the same time, from the pneumonic character of some of the sputa, there could be no doubt that a low form of pneumonia was present. We made a careful *post-mortem* examination, and the following are some of the details of it. We found a small quantity of fluid in the peritoneum, in the pericardium, and in the left pleura, where some old firm adhesions existed. The right pleura contained a large quantity of fluid. The heart was large, and weighed 2 lbs. avoirdupois. It was of pale colour, and very friable. The cavities were much dilated, and their walls thickened. The pulmonary artery and the aorta were much dilated. The valves on the right side were not diseased. The mitral valve was slightly thickened, but there was nothing to prevent its perfect closure. The aortic valves were also thickened by atheromatous deposit, but their surfaces were not rough. The aorta, at its commencement, and for some distance, was the seat of atheromatous and calcareous deposit. The dilatation of the aorta was so great, that the valves were incompetent to close the opening into the ventricle. The left lung was much engorged throughout, and contained much blood and frothy serum. The anterior part of its upper lobe was hepatized. The bronchial tubes were very red, and contained a large quantity of frothy mucus. The right lung was somewhat compressed by the fluid in the pleura. It was emphysematous in front. In its lower lobe there were several lobules which were the seat of extravasation of blood,—"pulmonary apoplexy." The kidneys were apparently healthy. The muscular fibres of the heart, when examined under the microscope, were found to have undergone extensive fatty degeneration; there were very few transverse striæ to be met with.

CHAPTER XVI.

GANGRENE OF THE LUNG — PHTHISIS, WITH FETID EXPECTORATION — BRONCHITIS, WITH FETID EXPECTORATION.

(CLINICAL LECTURE.)

GENTLEMEN, — Most of you, probably, recollect the case of John R——, who was in the hospital in March last (1866), and who suffered from symptoms which I told you, I believed, were produced by the existence of gangrene of the lung. The patient recovered from his attack, and, after remaining in the hospital for several weeks, was discharged. No opportunity, therefore, was afforded us of verifying the diagnosis which was made, yet I had no hesitation at the time the patient was with us, nor have I any now, in referring to his case as one of gangrene of the lung. Here is a short account of the case :—

John R——, 27 years of age, a labourer, a tall, strongly built man, was admitted into the hospital on the 2nd March, 1866. There was a history of hæmoptysis twelve months before admission, cough of three months' duration, loss of flesh, and occasional vomiting of food. Three days before he came to us he had coughed up some blood, and as the hæmoptysis continued, he applied to the hospital. I saw him for the first time on the 3rd of March. His pulse was then a little over 100. He had expectorated some dark, grumous, bloody matters, but I perceived no fetid odour. The front of both lungs was resonant on percussion, and the breath-sounds were good; the back of the chest was not examined. I prescribed some morphia and sulphuric acid, but he did not improve under the treatment; and on the 7th gallic acid was given, but it had no good effect; on the contrary, the patient was daily getting worse, and the quantity of dark-coloured matters expectorated was increasing. On the 8th the pulse was 120, and there were symptoms of exhaustion coming on. On the 10th I found him exceedingly weak, with an anxious expression of countenance, sunken cheeks, and a feeble pulse of upwards of 120. Moreover, he had brought up a large quantity of dark, bloody, fetid expectoration. I had now no doubt as to the nature of the case. On making a careful examination of the chest, we found deficient movement of the left side, crepitation in the left axilla, with dulness, bronchial breathing, and bronchophony at the left base. There was, moreover, prolonged expiration over the right apex. The heart-sounds were free from murmur. This examination of the chest convinced me that we had to deal with a very serious form of pneumonia, accompanied with gangrene. I at once stopped the sulphuric acid and morphia, and ordered a stimulating treatment, with as much nourishment as could be taken. The patient was put on a tablespoonful of brandy every two hours, with five grains of carbonate of ammonia, and some chloric ether, every four hours. On the 13th, the third day of the stimulating treatment, the pulse has 96. A large quantity of chocolate-coloured, tenacious, fetid matters had been expectorated. The patient said he felt rather stronger, and had rested better. The brandy was increased to 8 oz. daily, and the ammonia mixture was continued.

On the 15th, he expectorated about two pints of dark coloured fluid, and on the 16th, about a pint and a half; but the expectorated matters were of a lighter colour and less fetid than previously.

On the 17th he had passed a good night. The sputa were altered much in character, they were lighter in colour, less tenacious, and by no means so offensive. The following are the subsequent notes of the case.

18th. Sputa diminishing in quantity. Pulse 92, regular, and of good strength.

22nd. Improving. Expectoration scanty, and free from blood. Sat up for two hours yesterday. Takes nourishment well.

24th. Brandy diminished to six ounces. A pint of porter daily.

26th. Pulse 80; sits up every day for two or three hours. Sputa scanty, purulent, nearly free from fetor.

28th. The ammonia mixture was stopped, and two grains of quinine ordered three times a day.

April 7th. Since the last report the patient has been going on well. There is less cough and expectoration. The appetite is good; pulse 80. He was ordered an extra quantity of meat, with eight ounces of port wine instead of the brandy.

April 10th. There is some dulness at the base of the left lung, and slight crepitation; elsewhere the sounds are good.

14th. Quinine and iron ordered.

28th. Breath sounds normal over the base of the left lung. He was ordered cod liver oil in addition to the quinine and iron.

The patient remained in the hospital till the 17th of May, when he was sent to the convalescent hospital at Southport. On his return he came here, and appeared in excellent health.

Now, let me call your attention to the following case in which the expectoration and breath were extremely fetid, and there were very marked symptoms of prostration.

Patrick F——, a porter, of intemperate habits, 30 years of age, was admitted into the hospital on the 4th of March, 1867. He complained of cough and difficulty of breathing, and when we examined his chest, we found the physical signs of deposit at the upper part of the right lung, with bronchitic râles. He was ordered cod liver oil and iron. The pulse on admission was 96, but it fell after he had been in the hospital a few days; and on the 20th of March it was 88. About the end of the month, the patient began to expectorate very freely. The sputa were of a greenish colour, somewhat frothy and offensive. The breath was also offensive. You will recollect that I pointed out to you the peculiar odour of the breath and sputa. It resembled very much the odour of carious teeth. On the 30th March I ordered six ounces of brandy daily, and on the 9th of April I gave, in addition, some carbonate of ammonia. The patient was allowed a liberal diet. The symptoms continued without much change for about three weeks; the sputa were copious and fetid, and the breath at times very offensive. Further, the pulse became very rapid; there were profuse perspirations, and great prostration of the patient's strength. The area of dulness at the upper part of the right lung increased, and coarse moist râles were heard over a large part of the front of the lung.

Although the patient was taking a fair amount of stimulants, brandy and ammonia, I thought he was getting scarcely enough, and, accordingly, on the 23rd April, I increased the quantity of brandy to ten ounces a day, continuing the ammonia and good diet. An improvement almost immediately set in; the expectoration diminished in quantity, became less fetid, and, later, free from unpleasant odour. The pulse fell, the perspirations ceased, and the râles almost entirely disappeared, although dulness and deficient movement remained; the patient gained in strength and spirits, and you may recollect how much I was surprised at the condition of his chest, when I examined him on the 2nd of May. Here are the notes.

May 2nd. Dulness still persists at the right apex. There is more expansion of the right lung, but the movement is still deficient. The breath-sounds are improved, and are nearly free from moist râles. On the 9th of May we sent him to Southport.

Whether in this case any gangrene existed, or whether all the symptoms were produced by bronchitis, it is impossible to say; but, as in the case of R—— there was very severe constitutional disturbance, which yielded to the exhibition of stimulants and free nutrition.

Now let me call your attention to another case, which you have lately seen in the wards.

Charles N——, a sailor, 20 years of age, was admitted under my care, on the 2nd of September, 1867. He told us that he had suffered from cough, and pain in the left side, for about a month, having been previously in good health. He said he had expectorated dark coloured, fetid matter, but no blood. He had been obliged to give up his work for three weeks.

The patient was a strong-looking, well developed man. He had a quick and feeble pulse, and a somewhat anxious expression of countenance. His breath was offensive, and his expectoration of a dirty green colour, and very fetid. On examining his chest, we found some dulness, with crepitation at the base of the left lung; but we could find nothing abnormal in any other part of the chest—no evidence of tubercle, nor of bronchitis. I ordered an expectorant pill, two grains of quinine three times a day, and good diet. The symptoms continued for several days without any change, and, on the 7th September, I gave him eight ounces of port wine a day, and some chlorate of potash, with a chlorine gargle. This treatment seemed beneficial; the sputa diminished in quantity, but they still had the same character as at first—they were of a green colour, and at times very offensive, smelling very much like rotten apples. The pulse still kept up—I rarely found it much below 120—and the dulness and crepitation, although less, remained. The patient, however, improved in strength, he was free from fever, and from night perspirations.

On the 14th of September, he resumed the quinine, and on the 3rd of October I ordered him some cod liver oil, and, subsequently, I painted his chest with iodine. He improved under the treatment, and on the 24th October, his pulse was only 88. He was much stronger, had gained flesh, and expectorated much less. The sputa, however, were still fetid, and had not altered in their character; on two or three occasions a small quantity of blood was brought up. The man left the hospital on the 27th of October.

The most important point in connection with gangrene of the lung, next to its treatment, is its diagnosis; and this turns on the peculiar fetor of the breath and expectoration. Neither the general symptoms, nor the physical signs of the disease present any pathognomonic features. The prostration is, however, very great, and quite out of proportion to the physical signs. The pulse is very feeble and rapid; there is great dyspnœa; the face is sunken, and the aspect anxious. The fetor of the expectoration and of the breath is very peculiar, and you will recollect that, in all the cases I have referred to, I specially drew your attention to this circumstance.

But you may have fetid breath and expectoration, without gangrene of the lung, and, possibly, some might be disposed to doubt its existence, in the cases of Patrick F., and Charles N., but taking into consideration the severity of the symptoms in F.'s case, I am inclined to believe that a tubercular cavity had taken on gangrenous action; whilst in N.'s case, the dulness and crepitation indicated the existence of pneumonia, possibly set up around a gangrenous spot, or the offensive character of the sputa and breath may have been due to fetid bronchitis.

I have met with several cases, in which I have believed that there was nothing but bronchitis, or bronchitis with tubercular disease, and in which the expectoration and the breath have been very offensive. Such cases are not marked by severe symptoms, like those, for instance, which we had in R., but they are often very chronic, and very troublesome to cure.

In the treatment of cases of gangrene of the lung, or of fetid expectoration, you must be guided by the general condition of your patients, and you must give stimulants, tonics, and nourishment in proportion to the depression which exists. The medicines to be most relied on are carbonate of ammonia, quinine, chlorate of potash, and the mineral acids. In the case of R., I gave a considerable quantity of brandy, and in that of F., a still larger quantity. The treatment in both instances was successful, and this may encourage you to hope for a favourable issue, even in the most unfavourable-looking cases; for it scarcely seemed possible, at one time, that the former of the two patients could recover.

I must tell you that I think it highly probable that, in R.'s case, there were some tubercles in the lungs, for we had a history of previous hæmoptysis. In the case of F., I had very little doubt of the presence of tubercle at the right apex, and considering the man's broken down constitution, and his drunken habits, it was a matter of surprise to me that he did not succumb to his attack.

Chlorine has been recommended in the disease, but I have no experience of it, except when used as a gargle, and it has not seemed to me to have much influence in removing the fetor of the breath and expectoration. I see no objection to your using it internally, provided at the same time you give stimulants and tonics, but if it produce any nauseating effects, or interfere, in anywise, with the functions of the stomach, you must not persevere with it. It is of the first importance that you get your patients to take plenty of nourishment, and for this purpose you must try to keep the digestive organs in good condition.

Some differences of opinion have been expressed with reference to the blood-vessels implicated in gangrene; that is to say, whether it is the branches of the pulmonary artery, or those of the bronchial arteries, which are involved. I believe it is the former, and for the reason which I have given you in a previous lecture, viz., that they are the nutrient arteries of the air-sacs, and the sole vessels distributed to the parts especially implicated in pneumonia.

Gangrene of the lung is not a frequent termination of pneumonia. It is, perhaps, never a sequel of the acute form of the disease, and, probably, it very rarely occurs except in connection with some cachectic condition of the system. It may arise in the course of a chronic pneumonia, or where there is tubercular disease of the lung. It may occur, no doubt, without any previous inflammatory condition, and may set up, around its seat, a low form of inflammation, giving rise to all the physical signs of pneumonia. In such a case it is impossible to decide whether the pneumonia have preceded the gangrene, or followed it.

CHAPTER XVII.

PLEURISY—PLEURITIC EFFUSION.

(CLINICAL LECTURE.)

GENTLEMEN,—I wish to draw your attention, to-day, to some cases of pleurisy which I have had under my care, and to make them the subject of a few remarks on the nature of the disease, and the treatment which is most applicable to it. Pleurisy is, in my opinion, not quite as manageable a disease as pneumonia. It appears to me less easy of successful treatment, and, certainly, it is apt to leave behind it more permanent mischief than pneumonia. Probably, few cases of pleurisy occur without resulting in more or less extensive adhesions between the two pleural surfaces; and although these adhesions may not interfere seriously with the general health, still, they impair, to a certain extent, the function of the lungs. In pneumonia, however, there can be no doubt that, although there may be extensive effusion into the air-sacs, the whole of this may become absorbed, and the lung-substance may return to a perfectly healthy state. Again, pleurisy, more frequently than pneumonia, indicates the existence of some constitutional cachexia. It is very often of tubercular origin, and it is apt to occur in the early stages of pulmonary consumption. This fact should always be borne in mind in any case of pleurisy that may come before you, for it must necessarily influence your treatment very materially. Pleurisy very often sets in with but few marked symptoms, and runs a very insidious course, so that you may have patients presenting themselves to you for the first time, and you will find that there is a considerable quantity of fluid in one of the pleural cavities, and this, perhaps, without much increase in the frequency of the pulse, and without the presence of fever or pain.

Cases of acute pleurisy with high inflammatory fever, accompanied with rapid effusion, are by no means so frequently met with, either in our wards, or in private practice, as cases of acute pneumonia. In looking over my note-books I find few such cases recorded, although there are many where the disease has been of a more chronic and insidious nature.

Pleurisy and pneumonia are often combined. Indeed, probably most cases of pneumonia have more or less pleurisy associated with them; but the two diseases are essentially separate and distinct affections, and one may, and does exist without the other. The blood-vessels involved in the two diseases are different. In pneumonia, as I have already told you, it is the branches of the pulmonary arteries which are implicated; whereas, in pleurisy, it is, as far as the pleura covering the lung is concerned, the branches of the bronchial arteries which are involved. These (the bronchial arteries) are, I believe, the sole vessels concerned in the nutrition of the pulmonary pleura. I have found in lungs which have been the seat of pleurisy, in which there have been old deposits under the pleura, that, on injecting the bronchial artery, the vessels of the areolar tissue, into which the pleuritic effusion has become converted, have been filled with injection. Further, although in the healthy human lung, for reasons which I have explained elsewhere, it rarely happens that the vessels of the pleura become injected when the injection is thrown into the lungs through the bronchial arteries, yet, in some of the lower animals, injection of the pleura is easily effected through these vessels.

Before I speak to you of the acute forms of the disease, let me call your attention to some cases in which the affection has been more chronic; and first, I shall mention the case of a man who left the wards only a few days ago.

Mike C., a ship-fireman, was admitted into the hospital on the 19th October, 1867. The history he gave us was that he had had a severe cough, with dyspnœa, for about nine weeks, and that, for six or seven months previously, he had suffered from a slight cough. He had also had hæmoptysis, but he had not suffered from pain. He was a spare man, with an anxious expression of countenance. The pulse was 132, small, and rather jerking. There was no fever. On examining his chest, we found diminished movement of the left side, with dulness, and absence of breath-sounds and of vocal vibration, over the lower half of the left lung. The respiration was audible at the upper part of the lung, but it was faint. On the right side, the percussion and breath-sounds were good. The heart-sounds were not clear; the first sound being prolonged, and somewhat harsh, over the base, and in the course of the aorta, but there was no decided murmur. You will recollect that I pointed out to you all these symptoms, and that I said they distinctly showed the existence of fluid in the left pleura. I mentioned also, during the progress of the case, that I thought it highly probable that there was a slightly roughened condition of the base of the aorta, and that this would account for the somewhat abnormal first sound of the heart; and I was strengthened in this view by the circumstance that the arteries of the arm were firm and rather hard, indicating an atheromatous condition.

But to return to the pleuritic effusion, and the treatment we adopted for it. Bearing in mind the length of time the disease had probably existed,—nine weeks at least,—the weak condition of the patient, his rapid pulse, and the fear of a tubercular taint, I put him on a stimulating and tonic treatment. I gave him some bark and ammonia, with eight ounces of port wine daily, and meat; further, I ordered his chest to be well painted with tincture of iodine every day. On the 24th of October, his pulse had fallen to 94; and on the 26th, I substituted quinine for the bark and ammonia. On the 29th, the breath-sounds were faintly audible at the base of the left lung, showing that the effusion was becoming absorbed. I now ordered, in addition to the other measures, a table spoonful of cod liver oil three times a day. The iodine application was steadily persevered with, and on November 2nd, viz., a fortnight after the commencement of our treatment, we found evidence that the whole of the fluid had been absorbed from the pleura. The vocal vibration had returned, there was only slight dulness, and friction-sound was heard at the base of the lung. The slight dulness of the left side as compared with the right, I pointed out to you, was no doubt due to the thickening of the pleura, which always results, to a greater or less degree, in long-standing cases of pleurisy. There was another circumstance also observed, viz., a diminution in the capacity of the left side of the chest, as measured by the tape—the result of the collapse of the chest-walls during absorption of the fluid. From the date I have mentioned our patient improved, and he left the hospital on the 9th November. Thus you see that, with the aid of rest and the use of stimulants and tonics, and the external application of iodine, the pleuritic effusion was got rid of in this case. These were simple measures, but they were quite efficacious.

Now let me refer you to the case of Charles M'K——, 28 years of age, a tailor, a spare man, pale, and of somewhat strumous aspect, who was admitted into the hospital on the 15th May, 1867. He attributed his illness to having caught cold a fortnight before admission. We found him complaining of a good deal of pain in the right side, but the pulse was only 80, and there was but little constitutional disturbance. There was dulness on percussion over the lower half of the right lung, with ægophony and absence of breath-sounds. Further, there was deficient expansion of the side. We had no difficulty in diagnosing pleuritic effusion. I could not make out that there were any tubercles in the lungs, although, from the aspect of the patient, I suspected that he was of tubercular diathesis. I ordered him small doses of ammonia with morphia, three times a day, a third of a grain of morphia at night, a linseed meal poultice to the side, and six ounces of wine daily, with beef tea, etc. He continued this treatment until the 21st of May, when he had lost the pain in the side, but the physical signs continued without much change. I ordered him five grains of iodide of potassium, and twenty grains of acetate of potash, three times a day, and the wine was continued. He began to improve rapidly from this time. On the 25th, some tincture of bark was added to his mixture, and tincture of iodine was ordered to be painted over his chest daily. He was discharged on the 31st May, all evidence of effusion having disappeared.

Here are the notes of another case:—John R——, a joiner, 24 years of age, was admitted into the hospital on the 16th May, 1867. He said he had been troubled with a cough and shortness of breath for about a fortnight. He attributed his illness to having fallen on his side in October last. He was taken to a hospital, and remained there till February (four months), when he was discharged, quite well, and resumed his ordinary work as a joiner, which he followed up to a fortnight before he came to the hospital.

On examining him, we found he had a quick and weak pulse, 120. He was of somewhat strumous aspect, but there were no physical signs of tubercular deposit in either lung. There was, however, unmistakeable evidence of the existence of a large quantity of fluid in the right pleura. There was dulness, with absence of breath-sounds and of vocal vibration over a large portion of the right lung; in fact, in the sitting posture it was only quite at the apex of the lung that there was any resonance. I pointed out to you the circumstance that, by changing the posture of the patient, we altered the seat of dulness, thus proving, if any further evidence were necessary, that the dulness was due to the presence of fluid, and not of solid matter. We examined the urine, and found it had a specific gravity of 1·020, and was free from albumen. Now, let me impress upon you, in all such cases as that I am referring to, the importance of examining the urine, and of ascertaining, before you prescribe for your patients, whether they are suffering from albuminuria. You may perhaps be disposed, in treating some cases of pleurisy, to give mercury, for it is a remedy which is still recommended by some physicians. I shall, however, have to tell you bye and bye, that I have no confidence in it as a remedial agent, and that I rarely administer it, in pleuritic effusion, except as a purgative. You may, I say, be disposed to give mercury a trial; but you must never give it if your patient is suffering from Bright's disease. Therefore satisfy yourselves on this point before you prescribe. There can be no doubt that mercury is highly prejudicial to a patient who is the subject of organic disease of the kidneys; and I strongly advise you to abstain altogether from its use where that disease exists, whatever be the other affection from which your patient is suffering. And whilst I am on this point, let me remark that there is another drug, which, I believe, also exercises a very prejudicial effect in cases of Bright's disease, viz., opium, in all its forms. I am sure I have seen much mischief done in these cases by the exhibition of opium, or morphia. The drug is given with the view of procuring sleep, and it has, often, the desired effect; the patient sleeps, and sleeps soundly, but it is a sleep which hastens the fatal issue of the case. It is, probably, injurious by arresting secretion, and thus poisoning still further the blood, already loaded with impurities, from the imperfect action of the kidneys.

But to return to our patient. For the first two days I gave him carbonate of ammonia, and six ounces of brandy, daily. On the 18th May, his pulse had fallen to 104, and he had passed a good night; I ordered him five grains of iodide of potassium three times a day, and a blister to the side. He was allowed good diet. On the 22nd, he was attacked with diarrhœa, and we were obliged to omit the iodide of potassium, but it was resumed, with some bark, on the 25th, and tincture of iodine was ordered to be painted over the back of the right lung every day. I shall not trouble you with the details of the case. A steady improvement set in from the time the iodine was used, and on the 1st of June, the physical signs showed that a large portion of the fluid had been absorbed, and by the middle of the month all signs of its existence had disappeared. The patient remained some time in the hospital, and took cod-liver oil and iron. I suspected that some tubercles were present, although there were no positive signs of their existence.

But the cases, which I have drawn your attention to, came under our notice after the effusion had existed for some time; and I must tell you that this is, for the most part, the case in hospital practice. We do not often see our pleuritic patients at the onset of the disease. In private practice, however, we are called to them at an earlier period, and, occasionally, we have, in our wards, instances of severe acute pleurisy in the early stage. In such cases, the symptoms from which the patients suffer are often very severe, the pain is sometimes very acute, and the dyspnœa very urgent. The following cases will serve to illustrate the acute symptoms of the affection.

M.——, a domestic servant, aged 18, a somewhat pale anæmic-looking girl, was attacked with scarlet fever on March 16th, 1862. The fever was violent, but the rash was not well marked; and there was but little affection of the throat.

On the third day of the eruption, the urine was smoky, scanty, and highly albuminous.

On the eighth day the fever had subsided; the urine was almost free from albumen; and the patient was apparently progressing satisfactorily.

On the following day, she was attacked with inflammation about the throat and the glands of the neck, and the urine became more albuminous and smoky. She improved under treatment during the ensuing week, but the urine remained albuminous. There was anasarca, but to no great extent.

On the fifteenth day of her illness (viz., March 30th), she was seized with acute pain in left side, with great dyspnœa, and inability to lie down. The pulse was quick; the skin hot; and there was great thirst. She was ordered a glass of port wine (about two ounces) three times a day, and turpentine fomentations to the side.

On April 1st, the pulse was 110; respiration was painful and hurried; and there was still a sharp pain in the right side.

On the 2nd, there was dulness at the base of the right lung, with friction-sound higher up; the pulse was 100; and there was orthopnœa. Acetum cantharidis was painted over the side; she continued her wine, with a grain of quinine and three grains of ammonio-citrate of iron, twice a day; and she had a small opiate at night.

On the 3rd, she had passed a good night, was free from pain, and improving.

On the 4th, there was still dulness at the lower part of the lung; but there was no extension of inflammation. The urine was still smoky and albuminous, and of specific gravity 1.011. She was ordered ten grains of gallic acid twice a day, to continue her wine, and to have ten minims of laudanum at night.

On April 6th, she was sufficiently well to be removed from the house she was in, and was taken to the Northern Hospital. She steadily improved from this date; the chest-symptoms disappeared rapidly; and the fluid in the pleura soon became absorbed. She continued to take gallic acid in somewhat large doses, wine, and good diet. One application of tincture of iodine was made to the chest. The albuminuria was somewhat persistent, and did not altogether subside till after a treatment, partly with gallic acid, and partly with iron, of nearly seven weeks. She then left the hospital, quite well.

Michael L——, aged 35, a corn-porter, of intemperate habits, was admitted into the hospital on July 17th, 1860. Ten days before admission, he had slept in the open air on the grass. When he awoke he felt great pain in the left side, and this was followed by rigors and cough, which troubled him, more or less, up to the time of admission. He had slept badly, and had expectorated a good deal of white frothy matter; and on one occasion the sputum was red. No treatment had been adopted. When admitted he complained of dyspnœa, pain in the chest, and inability to lie on the left side; we found deficient movement of that side, dulness on percussion behind, reaching up to the level of the spine of the scapula, with absence of respiratory murmur, and of vocal fremitus over the same space, and distinct ægophony towards the base of the lung. On the right side the breathing was loud, and the percussion-sound normal. The pulse was 100, small, and compressible. He confessed to having been a "hard drinker;" he was moderately stout, but had a somewhat unhealthy aspect. He was ordered a purgative, and an eighth of a grain of tartar emetic, with acetate of ammonia, every four hours, four ounces of brandy daily, and beef tea.

On the 20th the pulse was 80, and the pain was diminished; the dulness was about the same; the brandy was reduced to three ounces, and a large blister was ordered to the left side. On the 21st the pulse was 76, and the breathing much relieved. The blister was discharging freely. On the 23rd the antimony and saline were omitted, and five grains of iodide of potassium, in bitter infusion, were ordered three times a day. On the 26th another blister was applied, and the brandy was reduced to two ounces; the dulness was diminished. On August 1st the respiratory murmur could be heard all over the left lung—distinctly, at the upper part, faintly, at the base. Chop diet and porter were ordered, and the brandy was omitted. On August 6th he was much improved. Slight dulness remained behind, and crepitation (crepitus redux) was heard at the base of the lung. He left the hospital on the 9th August.

Now you will have observed that, in neither of these cases, were any depletory, or severe antiphlogistic measures resorted to. In the first case the pleurisy was complicated with albuminuria, following scarlet fever. The symptoms were, however, very urgent, the pain was very severe, the sympathetic fever ran high, and the effusion was poured out somewhat rapidly. A moderately stimulating and tonic treatment, however, led to an early recovery from the disease.

The second was a distinct case of acute pleurisy, with, no doubt, slight pneumonia, for, at the termination, we had crepitus redux, so often heard in that affection. The attack was brought on by exposure to cold; but before the man came to us his disease had existed for several days, and some of the more severe symptoms had subsided. I used no depletion, and although I gave small doses of antimony for a few days, I also gave some stimulants. These were followed by iodide of potassium, and the patient made a rapid recovery. Thus you see that these acute cases of pleurisy, like acute cases of pneumonia, will often do very well without any depletion, and without the use of severe antiphlogistic medicines.

You will meet with cases of neglected pleurisy, where the effusion is so great that the whole of one side of the chest is absolutely dull on percussion, and no breath-sounds can be heard in any part. In such cases, if the pleurisy is on the left side, the heart will be pushed to the right of the sternum, and you will find it beating in one of the right intercostal spaces. If, on the other hand, the effusion is on the right side, the heart will be displaced upwards, and towards the left axilla. These are cases with which we sometimes have great difficulty—cases in which no medicines have any effect in promoting absorption of the effused matters, and in which we are obliged to resort to paracentesis thoracis, in order to relieve the patient of the fluid. You ought not, in my opinion, rashly to resort to this operation. It is your duty always to try the effects of treatment, for I have seen, in cases of very long standing, and apparently hopeless, a steady improvement, and, ultimately, perfect recovery from the persistent use of diuretics, and of external applications to the chest; care being taken, in the meantime, to support the patients' strength. The following are, I think, instructive cases:—

Antonio F——, 23 years of age, a sailor, was admitted into the hospital on June 18th, 1860. In the previous February he was laid up in hospital at Mobile with severe pain in the side and fever, which lasted for about three weeks. He remained in hospital for two months, and then sailed for this country, where he arrived about a fortnight before the date of admission mentioned above. During the first part of his voyage, he was able, partially, to do his work, but, subsequently, he was compelled to give it up. When admitted, he complained of dyspnœa, and pain in the left side on inspiration. On examination, the left side of the chest was found universally dull, both in front and behind, except in the supra-clavicular region. The dulness did not pass the median line in front. There was scarcely any perceptible movement of the left side during respiration; the side was full, but there was no particular prominence of the intercostal spaces. Over the left lung no respiratory murmur could be heard, except at the upper and anterior part, where it was faint; vocal fremitus was also absent, and ægophony could be distinctly heard both laterally and behind. The pulse was 84, and small. He was ordered two grains of iodide of potassium, with tincture of squills and juniper water, four times a day, and a croton oil liniment for the chest; ordinary diet, and porter.

On the 30th, the symptoms were about the same. Tincture of iodine was ordered to be painted over the chest daily.

On July 3rd, the respiratory murmur could be faintly heard at the apex of the lung behind.

On the 11th, the following was substituted for the tincture of iodine:—iodine ℈iss; iodide of potassium ʒ ij; water ℥ iij. He improved rapidly from the time this was freely applied to the chest.

On the 14th, the respiratory murmur could be distinctly heard at the upper part of the chest, both in front and behind.

On the 17th, the murmur was faintly heard at the lower part of the chest, and in the axilla, whilst the resonance was returning at the upper part. His health was excellent.

Further details are unnecessary. The patient improved daily; the external use of iodine was continued, and he took iodide of potassium internally, in bitter infusion. The following are the notes on August 1st:—Respiratory murmur heard all over the left lung; resonance natural, except at the extreme base, where there is slight dulness, and where the breath-sounds are somewhat faint.

He was discharged on August 3rd.

Thomas B——, 35 years of age, a seaman, was admitted into the hospital, under my care, on the 15th August, 1862.

The patient was a Swede, and could scarcely speak a word of English, so that we were unable to get any history of his case, and were obliged to rely on the general symptoms and physical signs in forming a diagnosis; we subsequently learned, however, that he had been ill for six weeks, with pain in the side.

We found the whole of the left side of the chest dull on percussion—absolutely so, except at the extreme upper part. No breath sounds could be heard, except at the apex of the lung.

There was scarcely any movement of the left side, on inspiration, and no vocal vibration could be felt on that side. The right side was resonant, and the respiration somewhat puerile over the right lung.

I ordered the patient ordinary diet, cod-liver oil, and croton oil liniment to the chest; and on the 23rd August, and again on the 28th, he was blistered. Up to the 30th of August, that is to say, for the first fortnight after his admission, no effect seemed to be produced on the disease, the dulness remained the same.

I now ordered tincture of iodine to be freely painted over the left side twice a day. I improved his diet, and gave him a pint of porter daily. I also ordered him two grains of iodide of potassium in infusion of gentian, three times a day. I, subsequently, on the 10th of September, increased the dose of iodide of potassium to four grains, and on the 25th, to five grains; on that day I find that the following note was made:—"Improving; the fluid is evidently becoming absorbed; the dulness of the left side is diminishing."

I find, after this, no note of his case till the 8th November, when there is the following report:—"He has continued the treatment last prescribed, viz., the iodide of potassium internally, and the tincture of iodine externally, the latter having been omitted from time to time, as it produced too much irritation. He has improved much in appearance, has no cough, and is stouter; there is only slight dulness at the lower part of the left lung, and the breath-sounds are heard nearly down to the extreme base." The treatment was continued, and the patient remained in the hospital till the 2nd of December, when he was discharged, well.

In the treatment of old-standing cases of pleuritic effusion, such as I have just referred to, I think that, unless the effects of the remedies we use, with the view of promoting absorption of the effused matters, show themselves speedily, we should resort to tapping. By continuing our remedies, we may lower our patients' health, and render them in a worse state to bear the operation. Further, and perhaps this is more important, time is allowed for the firm contraction of the matters effused on to the surface of the lung, and for the consolidation of the lung, which prevents its expansion when the fluid in the pleura is either absorbed or removed.

The external application of iodine is, I consider, one of the most valuable means we possess of producing absorption of chronic pleuritic effusions. The tincture of iodine sometimes fails to produce a good result, because it is not strong enough. You may then use some other preparation, such as the liquor iodi; that which I used, in the case of Antonio F——, answers very well.

You ought to be aware of one circumstance which occasionally happens, when iodine is freely applied over a large surface of the body, viz., the occurrence of a good deal of feverishness. I have often seen this symptom produced; the only thing you need do, is, to stop the use of the iodine for a day or two. It is in exceptional cases only that this symptom is met with, but it sometimes alarms the patient, and might also puzzle you, if you did not know its significance.

It is most important in the treatment of all cases of pleuritic effusion, to keep up the strength of your patients. There is a much greater probability of the fluid being absorbed when your patients are strong, than when they are weak; and, therefore, whilst you give your special remedies for promoting absorption,—and I think iodide of potassium is the best,—you must allow a good diet, and often some tonic, and even wine.

You must not despair too soon of getting these large pleuritic collections removed by absorption; but you must not, on the other hand, continue the use of your medicines, if you see indications of failing strength, and no diminution of the contents of the pleura; the time will then have arrived to relieve your patient by tapping.

I must tell you that I have no confidence in the powers of mercury to produce absorption of pleuritic effusions. I believe, as I have told you before, that, not only, does mercury possess no specific influence, as was formerly supposed, in subduing inflammation of serous membranes, and in promoting absorption of matters which are poured into their cavities, but that, if given to any extent, it produces, in most cases, positive harm. I have frequently watched its effects, and so satisfied am I of what I have just told you, that, except perhaps occasionally as a purgative, I never give it in these cases.

Blood-letting, whether general or local, is, I think, quite inadmissible in such cases of pleuritic effusion as I have referred to; and I doubt very much whether it is often desirable, even in the early stages, and the more acute forms of the affection. There can be no doubt that very great relief to pain follows the local abstraction of blood in many affections of the chest, but I have generally found that the same result may be brought about by the administration of opium. Whilst, however, I would deprecate any unnecessary abstraction of blood in the treatment of disease, I would express my dissent from the view that people will not, in the present day, bear the loss of blood as they formerly did. I believe that this opinion rests on no sound basis; and that the almost total disuse of blood-letting has not been brought about, in consequence of any alteration in the constitution of individuals, affecting their power to bear the loss of blood; nor yet, from inflammations having assumed a different type, or a less acute form, than formerly; but from a change in our views of the nature of inflammation, and of the therapeutics which it requires.

CHAPTER XVIII.

PHTHISIS—ITS NATURE AND TREATMENT.

(THE SUBSTANCE OF TWO CLINICAL LECTURES.)

GENTLEMEN,—There are few diseases which you will be called upon to treat more frequently than phthisis, or pulmonary consumption. It is unfortunately confined to no climate, to no class, to no age, to neither sex. It prevails, in varying degrees, both in high, and in low latitudes, and, in this country, it produces about one-eighth of the deaths from all causes. It used to be looked upon as an incurable malady, and when you consider what I have just told you of the proportion of deaths, which it occasions amongst us, you will at once see, not only how wide-spread is the disease, but how terribly fatal it is.

There can be no doubt that, of late years, the views of physicians with reference to the nature of phthisis, and the treatment by which it should be met, have undergone a material change—a change which has produced the most beneficial results. Many lives have undoubtedly been saved, and, in a still greater number of persons, life has been prolonged, by the measures, hygienic, climatic and therapeutic, which modern medicine has sanctioned. Much, however, remains to be done, for, as yet, we can only confess to a small measure of success, a measure, which, nevertheless, holds out hopes of greater success in the future.

You see, in the wards of this hospital, cases of consumption in all its stages. It is very rarely that we have not, at the same time, instances of the disease in the stage of deposit, in that of softening, and lastly, when the lung is more or less full of cavities. At the present moment, I have, under my care cases which afford illustrations of the physical signs, and general symptoms which characterize these several conditions.

It is not within my province as a clinical teacher, nor would it be possible, in one or two short lectures, to give a full account of what is known of the disease. My desire is, simply, to say a few words on the nature of phthisis, and, especially, to call your attention to the treatment which I think is most applicable to it, and to the results of my own experience of the value of hygiène, climate, and medicine in its management.

I shall purposely abstain from any discussion as to what tubercle is; great diversities of opinion exist on this point. It is sufficient, for my purpose, to impress upon you the fact, that phthisis is not a local, but a constitutional malady, either inherited or acquired; that it shews itself by a deposit of tubercle in the lungs, and that this tuberculous matter is, in my opinion, the result of an impaired, or defective nutrition.

The main causes which lead to the development of phthisis are especially at work in our great cities and towns, in the crowded and ill-ventilated houses, courts, and alleys, which are to be found there. It is the disease which, of all others, attacks the close-confined artisan, and the sempstress who sits, for many hours each day, breathing a vitiated atmosphere, and who rarely brings her lungs into free action. It is largely connected with an imperfect performance of the function of respiration, and this, perhaps, taken in its widest sense, is the main cause of pulmonary consumption.

Phthisis can be developed in some of the lower animals, by confining them in close, ill-ventilated chambers; whilst, on the other hand, it is rarely, perhaps, never met with in animals living in the open air, exposed, though they are, to all the vicissitudes of the weather.

Cold is not a cause of consumption. The disease is much more frequent amongst those who lead an in-door life, and are protected from the influence of cold, than amongst those who are exposed to the diminished temperature which an out-door life often involves.

Further, as I shall have to tell you hereafter, phthisis, far from being less prevalent in warm, than in cold climates, appears to be exactly the reverse. For it becomes more rare as we approach the poles; and in some tropical climates, it is, not only, very prevalent, but of a very malignant character.

Again, as cold is not a cause of phthisis, neither is damp; nor does it appear that a cold and damp climate, *per se*, is particularly favourable to the development of the disease.

I need not trouble you with statistics shewing the greater prevalence of phthisis in town districts, than in country districts. You will find such statistics in almost all your systematic works. Amongst the latest that have been furnished us, are those of Dr. Christison, in his address as President of the Health Section of the Social Science Association, in Edinburgh in 1863. Taking the mainland of Scotland, he found that the mortality from all diseases in town districts, as compared with country districts, was as 4 to 3; but the mortality from consumption was in the proportion of 333 for towns, to 186 for the country. In fact, the mortality was nearly double in the towns. Here are the figures. Dr. Christison says, that in Glasgow, 385 persons in every 100,000 of the population die annually of consumption; in Edinburgh, 283; in the North Highland counties, 179; in four lowland agricultural counties, excluding two towns, 138; in Fife, 125; and in Berwickshire, 104. Of this county Dr. Christison remarks, "in the county of Berwickshire, we have the most perfect example in Scotland of a population combining the richest agriculture with freedom from the deteriorating influences of mining, manufacture and large towns. None of its towns contain above 3,500 inhabitants; there is, I think, only one large factory in it, a paper manufactory, and there are no mines; here the deaths from consumption fall to 104 in the 100,000." This is one fourth of the proportion of Glasgow. These statistics for Scotland bear out those which have been drawn up for other parts of the empire, and point to the fact that consumption is essentially a disease of towns, and that one grand cause of it, as expressed by Dr. Christison, is "a conjunction of defective exercise and exclusion from the open air."

But, further, with regard to the influence of climate on the development of phthisis, a very remarkable statement was made by Dr. Christison, viz., that of all the inhabitants of Scotland, those who are the least liable to the disease are the inhabitants of the Western Isles, and that in the Isles of Lewis and Mull, phthisis is of very rare occurrence amongst the natives.

A tradition has long prevailed of the exemption of these Western islanders from pulmonary consumption, but Dr. Christison has given us some very valuable statistics on the subject; he applied to a very intelligent practitioner in the Isle of Lewis, Dr. Macrae; and the reply of this gentleman was to the effect, "that consumption in Lewis is almost confined to strangers temporarily resident there, and to natives who have resided and contracted the disease elsewhere, chiefly as domestic servants in the Southern towns of the mainland; and that the natives, who stick to the island, are exempt from the disease, except in a few rare instances, where it had been brought on under long privation of food and exposure to cold. Adverting to the defects in the register, and the jumbled mode of using the term consumption in the return, he adds, that he investigated the reported cases for the last three years in the Stornoway district, which contains a population of 8,500 inhabitants; that the total deaths were 444, or 1 in 61; that 24 deaths from consumption were registered; that every case had been seen at one period or another of its course by a medical man, so that he could trace it out accurately; that 8 of the 24 proved to have been bronchitis, 2 tabes, and 1 dropsy; that of the 13 true consumption, 5 were residenters from the mainland, and 4 native servants who had returned ill of the disease from service in Glasgow. Thus we have only 4 cases in three years among the true resident natives of the island, or 16 only to 100,000," the proportion for Glasgow being 385 per 100,000. "I have similar testimony," adds Dr. Christison, "from a very able authority in another island, Dr. M'Coll of Mull, who brings the experience of 33 years to the enquiry. He informs me that on his island, which contains 12,000 inhabitants, he has scarcely known consumption occur, except among immigrants bringing with them the constitution of the mainland, or natives who had gone thither early to contract it, but returned to die on the soil of their birth."

The strong testimony which we have here, to the exemption from pulmonary consumption of the inhabitants of these wet, cold, and stormy islands is very interesting and important.

In confirmation of the view, that immunity from consumption is enjoyed by those who live a life exactly the opposite of a town life, we have the testimony of Dr. Livingstone, the African traveller, who has informed us that the native tribes of South Africa, who know nothing of town employments and the confinement of civilized life, are free from consumption.

There are some further facts, in reference to the geographical distribution of phthisis, which I shall have to refer to hereafter.

The principal seat of tubercle in phthisis is the air-sacs of the lungs. The tubercular matter is poured out from the blood-vessels, and deposited in these cavities. It is also sometimes found in the smaller bronchial tubes. I have frequently examined lungs in the early stages of this disease, and have been able to pick out, with the aid of the dissecting microscope, small tubercular masses, having the shape and form of the air-sacs, to which the tubercular matter had been moulded.

The tubercular matter is described by some pathologists as infiltrating the areolar tissue between the air-sacs. But as no such tissue exists, it cannot be the seat of any deposit. Possibly, in some cases, the walls of the air-sacs may have tubercular matter in them; but I believe the main, and most frequent seat of deposit is the air-sacs themselves.

Whatever may be the tissue in which tubercle is deposited, it is in the apices of the lungs that, as a general rule, it first makes its appearance. This is an important fact in reference to the disease. The progress of the malady is, as a rule, from above, downwards; and a knowledge of this circumstance may be of material value in the diagnosis of some doubtful cases of disease.

There has been an opinion amongst pathologists, that the left lung is more prone to be first attacked than the right; but my own experience does not lead me to concur in this view. I find, on examining the records of my hospital cases, that the right lung has been more frequently the primary seat of disease. But we must not draw conclusions from limited experience. My belief is that, taking a large number of cases, it will be found that the right lung is quite as often the first to be attacked as the left.

Let me observe, with reference to the predisposing causes of the disease, that there is, perhaps, no occupation which specially and peculiarly gives rise to it. The man who leads an out-of-door life, or who is much in the open air, although exposed to great changes of temperature, is, the least of all persons, liable to phthisis. Whilst, on the other hand, those who follow an occupation, where are combined want of exercise, want of light, and want of pure air, although at the same time their food may be sufficient and good, furnish the largest number of victims to the malady. Whenever a predisposition exists, any circumstance which lowers the standard of health, which impoverishes the blood, or which gives rise to imperfect assimilation of the food, and, consequently, impaired nutrition, may determine the development of phthisis.

There is no period of life which is exempt from phthisis. It attacks both the young and the old. Youth and middle age are not the only periods of life which furnish victims to it. A large per centage of persons, who die over sixty years of age, are carried off by the disease, and the records of the Registrar-General show that, in relation to the number of persons living at the respective ages, the mortality from phthisis, between the ages of 15 and 70, does not vary as much as is, probably, generally believed.

According to the returns of the Registrar-General for the ten years, 1851 to 1860, the following was the relative annual proportion of deaths from consumption, to the number of people living at the respective ages:—

Under 5 years of age .	1 in 766
From 5 to 10 years .	1 ,, 1748

From	10 to 15 years	1 in 970
,,	15 ,, 20 ,,	1 ,, 337
,,	20 ,, 25 ,,	1 ,, 253
,,	25 ,, 35 ,,	1 ,, 231
,,	35 ,, 45 ,,	1 ,, 266
,,	45 ,, 55 ,,	1 ,, 288
,,	55 ,, 65 ,,	1 ,, 352
,,	65 ,, 75 ,,	1 ,, 504
,,	75 ,, 85 ,,	1 ,, 1174
,,	85 and upwards	1 ,, 2087

There is no temperament which does not furnish victims to consumption; nor can we say that there is any conformation of the body which is characteristic of the phthisical. I have seen men and women, with the best developed frames, and with the most ample chests, attacked with phthisis. You must not therefore be misled by the existence of these conditions, by the appearance of robustness in your patients, into imagining that they cannot possibly become the subjects of this disease.

There is, I believe, no climate in the world which affords absolute immunity from phthisis; no favoured spot in which, under certain habits of life, consumption may not become developed. But there is no doubt that the inhabitants of the high table lands of various countries have a remarkable exemption from the disease; and I have already pointed out to you that, even in the wet and stormy Western islands of Scotland, there is, according to Dr. Christison, but little consumption amongst the natives of the place, who, from the nature of their occupation, live much in the open air.

A very interesting discussion has taken place lately, at the International Congress of Paris, on pulmonary consumption, especially in reference to the prevalence of the malady in different climates. Dr. Lombard, of Geneva, exhibited a map, showing the altitude of different countries, with their effects on tuberculization. The higher the place, the less frequent was phthisis found to be; and, referring to the researches of Dr. Jourdanet of Mexico, Dr. Lombard stated that consumption is almost unknown in the high table lands of that country, whilst it is very common on the low grounds; and, further, that a similar state of things is observed in Switzerland. Dr. H. Weber has given, in the *British Medical Journal*, some very interesting cases, showing the curative influence of Alpine climates in the disease. All these facts are quite in accordance with my own experience of consumption, and with the remarkably beneficial effects of such climates in this, and other diseases depending upon defective nutrition. For cases of consumption in the early stage, and as places of residence for those who have a predisposition to the affection, I think the high lands of different countries are to be preferred.

But, setting aside, for the present, this question of elevation, I wish to impress upon your minds another fact, viz., that the countries, which are most exempt from the ravages of phthisis, are not the warm countries, but those in which the annual mean temperature is somewhat low.

Let me call your attention to a few facts in connection with this point. In Iceland, we are told, phthisis is extraordinarily rare; and the same remark applies to the Faroe Islands. In the country of the Esquimaux, and in the territory of Hudson's Bay, all travellers, says M. Bouchardat, agree as to the rarity of the disease. Again, the northern parts of Norway, of Sweden, the confines of Finland and Lapland, are also comparatively exempt from it. All Scandinavian physicians, in fact, agree that phthisis becomes less common towards the north, although the men, and especially the women, in those districts are lank, pale, and often ricketty. In St. Petersburg and Moscow, where the annual mean temperature is about 38° Fahrenheit; in Canada, and the northern parts of North America, the disease is said to be comparatively rare. In the Southern hemisphere the same state of things is said to prevail. Below Monte Video, we are told by Guibert, the frequency of phthisis diminishes, and further south still no examples of it are met with. On the western coast of the South American continent the same conditions are found.

Whilst, however, these colder climates present a comparative immunity from phthisis, there can be no doubt of its great prevalence in more temperate ones. It is said to cause 114 out of every 1,000 deaths in Vienna, 107 in Munich, 200 in Paris, 236 in London, and 135 in Berne. It attacks, we are told, a fourth part of the population at Marseilles, a sixth at Genoa, a seventh at Nice, an eighth at Naples and Philadelphia, and a tenth at Rome. It is common along the shores of the Mediterranean, in Malta, Corsica, and Sicily, as well as in Madeira.

According to Hirsch, phthisis is frequently met with in Arabia, India, Ceylon, and in the Indian archipelago; and it is very common and malignant in the South Sea Islands, in Australia, and in New Zealand. We are told by Couzier, that phthisis abounds in the Mauritius and the Isle of Bourbon. Along the coast of South America, along the sea-board of the Gulf of Mexico and of the Atlantic, in New Grenada, Venezuela, Guiana, and the Brazils, pulmonary consumption, according to Guibert, occupies the first rank in the pathology of these countries, and decimates the population, especially in the large towns.

In the Bermudas, and still more in the Antilles, it acquires a great malignity; and this fatal influence of hot climates has also been pointed out by physicians who have practised in India.

Thus it appears that pulmonary consumption is a widely spread, and very fatal malady, but that the climates which are the coldest enjoy the greatest immunity from it. How far this circumstance is the result of the mere climate, or how far certain conditions of existence have to do with it, I cannot now stop to enquire. At all events, its great prevalence in various climates disposes of the allegation that cold is a grand cause of the disease.

Density of population, and the overcrowding which necessarily results, are always attended with a high rate of mortality from phthisis; whilst, on the other hand, a sparse population possesses a great immunity from the disease. The fact that, in the colder climates which I have referred to, the population is less dense than in some of the warmer ones, may be an important element in the comparative freedom from phthisis which the inhabitants enjoy.

But there is the fact which I have alluded to above, about which I must say a few words, viz., the rarity of phthisis amongst the inhabitants of the high lands of different countries.

In Germany, phthisis is common enough in the large towns, but there is an almost complete immunity from it in the elevated districts; as in the Erzgebirge, between Saxony and Bohemia. In the Upper Hartz, at an elevation of six to seven hundred metres, out of 80,000 patients attended by Dr. Brockmann, only twenty-three tubercular cases were found, and of these, nine had brought the disease from other districts.

The same exemption prevails in the mountains of Hungary and of Styria, in the Carpathians, and in the valley of the Engadine, according to Brugger.

M. Lombard, of Geneva, says:—"If the low valleys, or the middle regions of the Alps present a large number of phthisical persons, the disease becomes more and more rare as we ascend the heights; so that, above 1,000 to 1,200 metres, only a few isolated cases are met with, and between 1,200 and 1,500 the disease disappears altogether.

According to Schnepp, phthisis is rare in the three communes of Laruns, Bages, and Eaux-Bonnes, situated at 521, 600, and 780 metres of elevation, in the lower Pyrenees. Again, in Africa, on the high plateaux of Abyssinia, phthisis disappears; and in India, according to various authors, it is infrequent, and even unknown in the high lands of the western Ghauts, and on the Neilgherry hills, at elevations of from 4,000 to 7,000 feet.

The same remark will apply to the elevated districts of America. I have already referred to the rarity of the disease on the high lands of Mexico; and now let me call your attention to a statement made by Dr. Archibald Smith, in the 41st volume of the 'Dublin Quarterly Journal,' with reference to the climate of Peru. In a valuable paper on the 'Climates of the Swiss Alps and the Peruvian Andes compared,' Dr. Smith remarks:—"Incipient tubercular phthisis, usually attended with more or less hæmoptysis, is one of the most common pulmonary affections known in Lima, and other parts of the coast of Peru. It is, besides, a disease almost certainly curable, if taken in time, by removing the coast patient so attacked to the open inland valley of Jauja, which runs from ten to eleven thousand feet above sea-level. This fact has been known and acted upon, from time immemorial, by the native inhabitants and physicians; and I have, myself, sent patients from the capital to Jauja in a very advanced stage of phthisis, with open ulceration and well marked caverns in the lungs, and seen them again, after a lapse of time, return to their homes, free from fever, and with every appearance of the disease being entirely arrested. But in many such instances it would, after a protracted residence on the coast, again become necessary to return to the mountains, to prevent the recurrence of the malady."

Again Dr. Smith observes:—"I may, however, say, in conclusion, that at the distance of twenty-five leagues from the town of Lombayeque, on the coast, is situated the Indian village called Cachen, on the lowest summit of the western Cordillera, as it approaches the shore of the Pacific, and the watershed falls equally to the east and to the west from this ridge. In the open air, at midnight, and in the dry season, the thermometer of Reaumur here fell to 4° (41° Fahr.), and rose to 7° (47.75° Fahr.) or 8° (50° Fahr.) by day, within doors. In the sun, the rise would probably be nearly twice as high as in the shade. Now this is one of the acknowledged health resorts for phthisis and hæmoptysis, contracted or developed on the coast of North Peru; and cases of recovery at this place are well accredited amongst trustworthy natives, who have themselves experienced its advantages. But the equable climate of the strath of Huanuco, with a night and day range of temperature from 66° to 72° Fahrenheit all the year over, is not favourable in similar cases, as I had the opportunity of testing; but at the colder elevations, up the sides of this Elysian valley, I have known decided benefit accrue to the phthisical and hæmoptic patient."

This immunity of the high table lands of different countries from phthisis, which is so prevalent, in the same countries, in the plains and great cities, and the curative influence of these mountainous districts, are very interesting and important facts. I shall not stop to enquire into the various reasons which have been assigned to account for them. In my opinion, the circumstances are due to the invigorating power of the atmosphere at these altitudes, its freedom from impurities, and also, which I consider of much importance, its dryness.

I have little doubt that, *per se*, the climate of these higher regions does not confer immunity from the disease, and that, under circumstances of dense population and overcrowding, we should see the disease largely developed. But this does not detract from the value of the facts I have referred to, which all tend to show the kind of climate, and habits of life which we should endeavour to select, not only for our patients predisposed to phthisis, but also for those who are already attacked by it. I have dwelt, somewhat at length, on this subject, because my own observations of the disease, and of the effects of remedial measures for its relief, lead me theoretically to conclusions, which the statements I have referred to bear out.

Of the two forms of phthisis, the inherited, and the acquired, there can be no doubt, I think, that the former is the more difficult to treat successfully, and that it offers less chance of a favourable result from the use of therapeutic measures. When consumption arises as the consequence of an inherited cachexia, and especially when it occurs in those who, from their birth, have been surrounded by good hygienic conditions, and have been furnished with all the comforts, and, possibly, all the luxuries of life, the ordinary hygienic, climatic, and therapeutic measures often fail to have the beneficial effect which so frequently characterizes their use, when the disease has been acquired from following some unhealthy occupation, or from living in the midst of insalubrious conditions. In the latter case, the withdrawal of the patient from his occupation, or removing him from the close and confined atmosphere in which he has been living, allows the natural strength of his constitution to come into play: and if this be assisted by ordinary measures, even if he be in no better air than that of a hospital, a rapid improvement often takes place.

But in the cases of inherited disease, to which I have referred, a mere change of air is often not enough. It constitutes, in fact, but a small change for your patients, a far less change than that which you adopt in the over-worked artisan, or the tired-out sempstress.

I must tell you that I look upon phthisis as a curable malady; and, I think, the opinion that it is so is now pretty general in the profession. But whilst I say this, I must add that, assuredly, we cannot count our complete recoveries by large numbers; and that, if phthisis is to be cured, it must be taken at an early period of its existence, and persistently treated. Many a case has been benefited for a time, the disease has been apparently subdued, when a return to old habits, to old occupations,—to the original causes, in fact, of the malady, has led to a re-appearance of the affection, in a severe form, and to a fatal issue. You may learn an important lesson from this,—not to boast too soon of your cures, and to insist on the importance, not only of prolonged treatment in all cases of consumption, but of your patients leading such a life, as will enable them, if possible, to avoid the causes which are likely to bring their latent disease into activity.

There can, I think, be no doubt that modern medicine has done much for the phthisical patient; that, under the system of treatment which now prevails, the disease runs a less rapid course, and recovery from it is far more frequent, than was formerly the case. I can bear strong testimony to the value of the mode of treatment, which has of late years gained favour with the profession.

I shall not dwell on the symptoms or physical signs of the disease, for you will find all these detailed in your systematic works; but I shall pass on, at once, to say a few words in reference to the principles of treatment.

There is no disease in which it is more important to insist on a strict hygiène than in phthisis; and, by this, I mean everything that relates to diet, exercise, ventilation, clothing, the action of the skin, etc.

The diet of a consumptive patient should be nutritious, easy of digestion, and more or less varied. The quantity of food taken should only be limited by the powers of digestion, and the number of meals must depend, not upon any strict rule, but on the circumstances of each patient. It is far better, for some people, to take four, or even five small meals a day, such as the stomach is able to digest without any sensation of weight or pain, than two or three heavier meals, followed each time by a sensation of oppression. When phthisis has only slightly advanced, if your patient can eat and digest well, there is good hope of his recovery; if he cannot, there is little or none. One great value of the various tonics we administer, and of the change of air we prescribe, is in the effect they often produce on the digestive organs, in improving the appetite, and in enabling the system to assimilate more food. This latter point is of much importance in all diseases of a nature similar to phthisis. There is no doubt that, with many persons, there is a great waste of food, that a considerable portion of what is taken is either not properly digested, or, if digested, is not assimilated. Not only do people differ much in this respect, but the same persons differ much at different times, and under different conditions; and one great benefit, often derived from the measures you recommend to your patients, is, that they are able, not only to eat more, but that they assimilate a larger proportion of their food, and thus rapidly gain flesh.

The diet of your phthisical patients should be, by no means, restricted, and may include the ordinary articles of food — meat, fish, fowl, game, etc. Bread and milk form an excellent diet for the consumptive, and are often much liked by them.

To a liberal diet should be added some alcoholic stimulant, wine, beer, or porter, the selection being made according to the condition of the stomach. If the digestion be weak, one of the light wines of France or Germany will probably be best borne— champagne, given with or without water, hock, claret, or burgundy. But some patients are able to take the stronger wines, port or sherry, and are often much benefited by them, especially by the former.

And now let me say a few words as to the air which your patients should breathe, and the temperature in which they should live. As a rule, phthisical patients, in whatever stage of the disease they may be, bear ventilation well. It is the hot, close, confined atmosphere which distresses them, aggravates their cough, and prevents them from sleeping. To the patient whose disease is incipient, fresh air is the main element of cure; to the patient whose disease is advanced, it is often the main element of comfort. Draughts are less dangerous to the consumptive than foul air, but draughts should be avoided.

It is not wise, speaking generally, to allow a consumptive patient to sleep with the bedroom window open; but the ventilation of the apartment can be easily secured by opening a window in an adjoining room or passage. The inflammatory attacks, which come on in these cases, yield more readily to a tonic and stimulating treatment, in which pure air forms a part, than to any other; and you may often send a patient who is suffering from bronchitis, or even from pneumonia, to spend a large part of the day in the open air, either on sea or land, with the best possible results. You may have some difficulty in arranging these matters for your patients, for there is still much ignorance amongst the public in regard to the importance of fresh air, both in health and disease; but a little tact and discretion will generally enable you to remove existing prejudices, and to get your directions carried out.

One word as to the temperature of your patients' rooms. You need not be afraid of letting them breathe cool air. The temperature of their rooms, whether by day or by night, should never be high. From 60° to 65° Fahrenheit, is a good temperature for a sitting-room, and the bed-room should be cooler.

Clothing is another very important matter in regard to your phthisical patients. Some woollen garment should always be worn next to the skin. You will find a great neglect of proper clothing amongst all classes of people, independently of the exigencies of fashion. In all climates, where there are sudden and great variations of temperature, some substance should be worn next to the skin which tends to preserve the body from these changes, and wool fulfils these requirements.

It is very desirable that a good action of the skin should be kept up, not only by wearing proper clothing, but also by daily ablution. You may safely allow your phthisical patients to use cold or tepid sponging of the whole surface of the body, daily; and this application of water to the skin should be followed by strong friction.

I have said nothing as to the exercise which your patients should take. All violent exercise must be avoided; walking, riding, light gymnastics, yachting, carriage exercise —all these are good, and all may be used in their turn, according to the condition of your patient; the more you bring the lungs into action, in cases of incipient phthisis, I believe, as a rule, the better; and this is no where so well accomplished as on an elevated spot, in which the air is rarefied and dry. The only thing you have to guard against, in reference to exercise, is, over-fatigue—that amount of fatigue which may prevent your patient, when he returns home, from either eating or digesting his food. It is obvious that this can only result in mischief. And, in regulating the exercise of the phthisical, you must bear in mind that their disease is essentially one of debility, and that, although slight fatigue may do no harm, great fatigue tends seriously to impair their vitality. The same remark will apply to all mental exertion and anxiety. We all know how great is the strain on organic life of mental labour, how, even when the organs are sound, it often produces serious functional disturbance, and lays the seed of future structural disease. And the same with anxiety of mind; the digestive process becomes deranged, assimilation is impaired, and a faulty nutrition is the result. How often do we see that the hard-worked professional man, threatened with organic disease, or perhaps already attacked by it, if he can be induced to give up his labours, and thus get rid of the anxieties, worries, and mental exertion incident to his occupation, begins immediately to improve in health, and, perhaps, ultimately shakes off his disease.

And now let me suppose, Gentlemen, that you have to deal with a case of phthisis in its early stage, when there is evidence of commencing failure of the general health, and of a deposit of tubercular matter in one of the lungs. What are you to do? I will suppose, that the circumstances of your patient are such, that he can adopt any plan you think best for him. You must see that the hygienic measures, which I have detailed, are strictly carried out, and you must direct your patient to be as much as possible in the open air, whenever the weather is fine, and moderately warm. If he live in an inland district, a change to the sea-side may be very beneficial; or, if he live at the sea-side, a change to a dry, elevated, inland spot may be advantageous. Patients do very well in this country during the summer months, from June to the middle of October, and the places which, in my opinion, are most likely to benefit them, are such as are tolerably high, dry, and bracing. The beneficial effects, which some phthisical patients have derived from visiting the mountainous parts of Switzerland, afford satisfactory evidence of the value of such a climate. I believe the worst place to which you could send your patients suffering from phthisis, is one in which the air is moist and warm. The depression, which such a climate produces, gives rise to languor, want of appetite, and an unwillingness, and even an inability to take exercise—circumstances which cannot be otherwise than injurious. On the other hand, I believe, that the climate most favourable to the consumptive is that, in which the air is dry, the land tolerably elevated, and the temperature moderately warm. Good summer residences are to be found in many parts of our own country, on the hills of Scotland and of Wales, in various parts of our sea-coast, and in the higher inland districts of England.

But what are you to do with your patient during the winter months, from October to the end of May? I think one of the best things you can recommend to him, is, to go on a long sea voyage. I have seen great benefit derived from this, and I am in the habit of recommending patients to take a voyage to Australia, in the winter months. During the past few years, several of my patients have followed this advice, and I have had the opportunity, not only of examining them on their return home, but of watching them afterwards. I have sent patients to Australia, who, at the time they left England, were suffering, some, from phthisis in its first stage, and others, from phthisis in its second stage. In some cases there was dulness on percussion, with deficient movement of one side, and prolonged expiration; whilst in others there were indications of commencing softening. The cases I have sent have all done well. In none of them was the disease far advanced; in none was there marked failure of the general health, although in all the strength was beginning to be affected. In one case, that of a gentleman between 20 and 30 years of age, there had been, on more than one occasion, severe hæmoptysis, and from the circumstances of the patient's life, his previous habits, etc., a rapidly downward course was much feared. He returned, however, from his voyage greatly improved, and the following year he went back to Australia to settle. When I last heard of him he was in good health.

There are certain precautions to be taken in reference to the voyage. The patient should leave this country not later than the end of October, and he should not return to it earlier than the middle, or end of May. May is sometimes a very cold month in England, and it is well that no risk should be run by coming back too soon. There is yet another circumstance I must mention in reference to the voyage; the return home from Australia should not be by the ordinary route, viz., by Cape Horn. I have known instances where patients have kept quite well up to the time they reached Cape Horn, on their homeward voyage, and having taken cold, in encountering the fogs and bad weather so frequently met with there, have never recovered from its effects. On leaving Australia, the return to Europe should be made by Panama. There is now a line of steamers running between Panama and this country. It is a good plan to send your patients by a slow vessel, in order that they may be longer at sea; but the accommodation which the vessel affords is a circumstance to be taken into consideration.

This plan of sending your patients on a voyage to Australia and back, during the winter and spring months, is, I think, in properly selected cases, the best you can adopt; but there may, and often will be circumstances which will render your patient unwilling, or unable, to follow such a course, and then you must select some spot for a winter residence, either in this country or elsewhere. There are places on our Southern, and also on our Western coast, which possess many advantages. The main object you have in view, is, to put your patients in such a climate that they may be able to spend a good deal of time in the open air. The line of the Italian coast has long enjoyed a high reputation, as a winter residence for the consumptive, and there can be no doubt that it offers great attractions; but, at the same time, I think there are places in our own country, which, in a large number of cases, are equally good as places of resort.

Madeira used to be a favourite spot to send consumptive patients to in the winter; but experience has shown that its climate possesses no peculiar advantages, and of late years it has been very much abandoned. To whatever place you send your patients, you must impress upon them the necessity of following out the hygienic rules which you may have laid down for their guidance. Some people seem to imagine that the climate alone, especially if it be a Southern one, will do everything for them; that, arrived there, they may dispense with those precautions which they adopt in their own country; and thus they are led into excesses, or imprudences, which prevent any beneficial effect, which, under a proper system, they might derive from the climate itself.

There is one thing I would warn you against, viz., sending your patients away from home, when you feel morally sure that no real benefit is likely to ensue. It is sometimes difficult to dissuade patients from undertaking, and their friends from allowing them to undertake, a journey to some spot, which, if they could reach it, they imagine would effect wonders in their case. They will often suggest a removal to some Southern climate when they are struggling in the last stage of the disease, and when you know that it is only a question of weeks, or of months at the outside, during which life can be prolonged. If you try to convince the patients, or their friends, of the uselessness of the plan which they propose, and to which they cling as a last hope they are apt, sometimes, to think that you take a too desponding view of the case, and to seek advice elsewhere, advice which may coincide with their own wishes, and the unfortunate patients may be transported to some distant spot, only to die as soon as their destination is reached, or to linger for a short time, away from all the comforts and conveniences of home. It becomes, sometimes, a hard matter to speak decidedly on the futility, to say the least of it, of removing patients, under circumstances such as I have described; nevertheless, it is often our duty to do so, although we thus seem to cut the last frail thread by which they cling to life.

But, where the disease is not far advanced, it is always desirable that patients should be removed, if possible, to some properly selected spot. either in this country, or further south still, during the winter months. It is, however, quite certain that, in the early stage of the disease, some cases will do quite well in an ordinary country district, even in the North of England.

In the autumn of a few years ago, one of my patients brought his sister, about thirty years of age, who was living in Yorkshire, on the borders of Cumberland, to see me. She was suffering from phthisis, in the first stage. There was loss of flesh, and the appetite was impaired. The pulse was quick, she had slight hæmoptysis every morning, and her strength was beginning to fail. She remained in Liverpool about six weeks, and, with the aid of cod-liver oil and iron, she steadily improved and gained flesh. I recommended a voyage to Australia, or, if that were not possible, that she should go to the south of England during the winter months. I gave her directions what to do in case she decided to remain in Yorkshire. In the following June she came to me again. She had remained in Yorkshire during the winter. I carefully examined her chest. I found evidence of improvement. There was less dulness, and more expansion of the affected lung. The patient had gained flesh, and she considered herself quite well. She had continued to take, from the time I previously saw her, with occasional intermissions, cod-liver oil and iron. She had lived well, and had been careful not to go out, except when the weather was fine. Had this patient gone, either to Australia, or to the south of Europe, or to the south of England, and returned to me in the condition I found her after she had wintered in the north, I should certainly have considered her a good instance of the beneficial effects of change of climate. I might refer you to other cases of a similar kind, but this one must be sufficient. You must not draw too hasty a conclusion from it; but, at the same time, it will shew you that it is not always necessary to remove your patients to the south at the approach of the winter season, and that they will sometimes do very well in our northern counties.

I now come to speak of the medicines which may be of use in the treatment of phthisis. You will have observed that I do not prescribe a great variety of remedies to the patients who are under my care; that the list, in fact, is almost reduced to cod-liver oil, quinine, and iron. These are, in my opinion, the chief medicinal substances—if, indeed, we can call oil a medicine—by the exhibition of which we can influence the development and progress of tubercle.

There can be no doubt as to the value of cod-liver oil. It is quite unnecessary that I should say a single word on that point; nor, indeed, shall I attempt to explain to you its mode of action. You have seen it used so frequently, and with such decided benefit, that you cannot hesitate for a moment, as to the propriety of giving it in cases of consumption.

It is, as a general rule, well borne by the stomach, and it is remarkable how some patients get to like it. Unfortunately, however, some people cannot take it; but still, you must not readily allow them to give it up its use, but you must try to find some vehicle in which it will be tolerated. Some persons take it best with warm milk, others with orange wine, some again with a bitter infusion, etc.

I very frequently prescribe quinine, in small doses, for my phthisical patients, and as a tonic I believe it is very useful. You need no particular directions for its use. You may give it with a bitter infusion if you like, in conjunction with some mineral acid.

Iron, in some cases of phthisis, is of especial value; of greater value than quinine in the cases which it suits, but it is not applicable to all cases. There are some in which it seems to do no good whatever, and in which, instead of the patient gaining flesh under its use, he either remains stationary or retrogrades. There is, I think, no better preparation than the tincture of the sesquichloride; but you can use other forms of the mineral if this particular one should not agree with your patient.

Glycerine I have occasionally given to children, when I could not get them to take cod-liver oil, and it has appeared to do good. My experience of its use is, however, limited, and I should not advise you to trust to it, unless you cannot get the oil taken.

Sugar is spoken of as useful in phthisis, and, as a carbonaceous substance, and therefore likely to produce fat, I should say, theoretically, that it might be of service in the disease. But I have no experience of it. I have never treated a case, solely, by administering large quantities of sugar.

I attach some importance to the use of mild counter-irritants in the earlier stages of phthisis, such as tincture of iodine, painted over the chest; but, I think, that a powerful irritant is not desirable, unless there be some inflammatory symptoms calling for it. The tincture of iodine is a very safe application, and, if continued for some time, it keeps up a slight external inflammation, which is, in my opinion, all that is necessary.

Let me say a few words about cough mixtures. As a rule, I think, the less you give them to your phthisical patients the better. Small doses of morphia, combined, or not, with chloric ether, are often of use in allaying a cough, and thus enabling the patient to pass a comfortable day, and to sleep well at night; but as the great safety of your patient depends on the integrity of the digestive organs, and their power of digesting food, you must, as far as possible, avoid giving anything which may interfere with their action in this respect. Now, most of the substances, of which cough mixtures are usually composed, are more or less nauseating, and, for that reason, objectionable. You must not be led away, in the treatment of your phthisical patients, from the main object you have in view, viz., the improvement of the general health, by the desire which they may express for a remedy for this, or that particular symptom, which, although it may be somewhat distressing, and might be temporarily soothed by the exhibition of opiates, etc., will be most effectually relieved by constitutional treatment. Counter-irritation is often much more effectual in relieving a cough than any medicine you can give internally.

Let me caution you against interfering too much with your patients' bowels by the administration of, so-called, alteratives. Of the value, in some cases, of some of the medicines to which the name, "alteratives," has been given, there can be no doubt; but do not be led into the mistake that, whenever there is some little disturbance of the bowels of your phthisical patients, some little dyspeptic attack, some, so-called, "biliousness," or a furred tongue, it is necessary to give a dose of blue pill, or some other mercurial. As a rule, you should always avoid giving mercury to a consumptive patient; but, in some cases, it is necessary to give one or more small doses. In all diseases of impaired or defective nutrition, mercury acts prejudicially; therefore do not give it, without due consideration, in phthisis. The compound rhubarb pill, or the compound colocynth pill with henbane, will usually fulfil all the necessary requirements, and may be given with safety. But a change of diet alone may suffice, and you should avoid, as far as possible, giving any medicine of the kind I have mentioned to your patients.

One word in reference to the influence of hospital residence. A hospital is one of the worst places for showing the good effects of treatment in consumption, and yet you must have observed that many of the patients whom we admit derive great benefit during their stay. The fact is, that most of those who come here, suffering from the disease, have been previously living under bad hygienic arrangements, and the change for them is one, from a bad, into a comparatively good air. Moreover, they get a cessation from labour, good diet, and good nursing. It is no wonder, therefore, that they begin to improve at once; but, unfortunately, after a few weeks of steady improvement, they very frequently come to a stand-still. The hospital has done all it can for them, and their cure, if cure can be effected, must be carried on elsewhere.

I have not spoken of the various complications which often arise in the progress of consumption, for my object has been to refer to the general principles of treatment for the constitutional malady, and not to point out special remedies for particular symptoms. I would strongly impress on your minds the importance of an early recognition of the disease, and therefore that you should carefully study the physical signs by which it is characterized. If your treatment of phthisis is to be successful, it ought to be commenced early, and your patient should be at once removed from those conditions of life which are favourable to its development.

Since the lectures which form the present chapter were delivered, attention has been strongly directed to the possibility of inoculating tubercle, especially by the researches of M. Villemin,* who claims to have made the discovery that tubercle is inoculable. M. Villemin, according to his own observations, has succeeded in producing tuberculosis in rabbits, by inoculating them with tubercular matter taken from man; and, further, he has produced the disease in other rabbits, by the inoculation of tubercle taken from the cow. Again, he has taken tubercle from a recently killed tuberculous rabbit, and, with it, inoculated other rabbits, and these have been found tubercular. He has, also, succeeded in producing tuberculosis in guinea-pigs and dogs, by inoculation from the human subject. The experiments which he performed are very carefully detailed, and he considers that they are "sufficient to establish that tuberculosis is inoculable from man to certain kinds of animals, and from animals to other animals, of the same species, and of different species." Some experiments, bearing on this subject, have been performed by Dr. Marcet, and have been communicated to the Royal Medical and Chirurgical Society of London. These experiments consisted in inoculating rabbits and guinea-pigs with the sputa of phthisical patients, and in one instance with the pus of empyema. Several of the animals, we are told, when examined after death, exhibited tubercles to a greater or less extent; and not only were they found in the animals which were inoculated with phthisical sputa, but also in the one inoculated with the pus of empyema. I must refer to the work of M. Villemin, and to the paper of Dr. Marcet, for further information on these points. The facts which are there detailed are very interesting, and highly important; but before we can admit, as an established truth, that tubercle is inoculable, we must have further proof that the deposits produced by these experiments were really tubercular. The circumstance that in the experiment of Dr. Marcet, which I have referred to, the so-called tubercles were produced by the inoculation of pus taken from the pleura, seems to me to be capable of affording a different explanation to the whole series of experiments, than that which has been drawn from them.

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* "Etudes sur la Tuberculose." Par J. A. Villemin. Paris, 1868.

CHAPTER XIX.

RESEARCHES ON ASPHYXIA (APNŒA) — WITH OBSERVATIONS ON THE EFFECTS PRODUCED BY THE HOT BATH IN ASPHYXIATED ANIMALS, AND ITS USE IN THE TREATMENT OF SUSPENDED ANIMATION.

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Although numerous investigations have been made with reference to the nature of asphyxia, and the influence of various modes of treatment in restoring suspended animation, I am not aware that any previous observers have directed their attention to one of the points, which it was the object of the experiments referred to below, more particularly to inquire into.

Of the intimate pathological changes which take place in asphyxia, our knowledge is still imperfect, and we are not yet agreed as to the best modes of treatment in the more acute forms of the affection. The various societies, which direct their efforts to the saving of human life, issue rules for restoring suspended animation, of the most opposite character; for whilst some recommend the use of the warm bath, others expressly forbid it, as not only worthless, but dangerous.*

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* Since this paper was written (1861), the societies referred to have, for the most part, modified their directions, and have recommended Dr. Marshall Hall's and Dr. Silvester's methods of performing artificial respiration; further they have, at least in some instances, prohibited the use of the hot bath.

Physiologists concur as to the order in which the arrest of the vital actions takes place when the entrance of air to the lungs is cut off. They agree that the function of respiration is stopped, and that animal life (so called) becomes extinct, before the cessation of the heart's action. With regard, however, to the period during which the latter organ continues to contract after asphyxia has commenced, opinions do not so fully accord.

The success which, in many instances, has attended the efforts to restore animation, where the function of the lungs had only just ceased, encourages us to hope that, in all cases in which the action of the heart is still going on, we may, by an improved method of performing artificial respiration, restore life; and the want of success (except in rare instances), in those cases where respiration had ceased for some time, and where, probably, the heart was no longer beating, should act as a warning to us not to lose a single instant in endeavouring to promote the respiratory changes, to which alone we must look for a favourable issue to our efforts.

It is a matter of great importance, in connection with the treatment of asphyxia, to ascertain the period after the cessation of the respiratory changes during which attempts to reanimate are likely to be successful; and this must depend on the time the heart continues to beat. Further, it is also important, considering the differences of opinion which now exist on the subject, to estimate aright the value of the hot bath as a remedial agent in the affection; and to do this it becomes essential to ascertain its effects on an animal when in an asphyxiated condition.

With the view of throwing some light on the points above alluded to, I instituted a series of experiments, the definite object of which was to answer, as far as possible, the following questions:

1. How long does the heart continue to beat in asphyxia?

2. What are the effects of the hot bath on asphyxiated animals: first, after all respiratory movements have ceased, and are not re-excited; secondly, when respiration has been re-excited and is being feebly carried on?

My experiments were made on animals asphyxiated by submersion; but as the morbid changes which take place in asphyxia are the same by whatever means it may be produced, I think it may be fairly assumed that the remarks I shall have to make and the inferences I shall draw, a far as they may be correct, will be generally applicable to all acute forms of the affection.*

With regard to the first of the above questions, it is obviously very difficult to come to anything like a correct conclusion. As far as man is concerned, it is indeed impossible to do so, and even with reference to the lower animals it is very difficult; a large number of experiments becomes necessary in order to obtain an approximation to the truth. The principle generally admitted is that "in asphyxia the movements of the heart cease in a few minutes after the cessation of the functions of animal life;" but the occurrence of certain facts, such as the recovery of individuals who have been under water for several minutes, would lead us to doubt the truth of the assertion. The second question seems more readily answerable, but yet no direct experiments bearing on it appear to have been performed previous to my own, some of which were made four years ago.

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* The mode in which apnœa is produced has a material influence in reference to the probability of suspended animation being restored. Thus it has been proved, that apnœa from ordinary suffocation, lasting for a given time, may be recovered from, whilst apnœa from drowning, lasting for the same time, is usually fatal. The reason is obvious. In the first instance, the lungs are free from accumulations; in the second, they are more or less loaded with water and frothy mucus. As all my experiments were made in reference to apnœa produced by submersion, my remarks must be considered as applying specially to that form.

The experiments, given in the tables below, were performed on warm-blooded animals — dogs, cats, and rabbits. Those referred to in table No. 1 were conducted in the following manner: — The animals were plunged into water which varied in temperature from 40° to 50° Fahr., being a few degrees below that of the surrounding atmosphere, in one instance the temperature being reduced to 36° artificially; they were kept completely under water from the time of their immersion, and when removed (after every external symptom of life had disappeared), were, either at once or after the lapse of a stated time, opened by the removal of a portion of the anterior walls of the thorax; the movements of the heart were thus observed without the pericardium being disturbed. Some of the experiments, viz., 9 and 10, 11 and 12, 14 and 15, and 17 and 18, were parallel experiments, *i. e.* the two animals of each experiment were of the same age and size, were submitted to exactly the same process of drowning, and were opened at, as near as possible, the same time, the difference between the two being that one was put into the hot bath for the period mentioned, the other was left on the table exposed to the atmosphere during the same time. The experiments 20 and 21, and 22 and 23, were also parallel experiments; solely directed, however, to show the pathological effects of the hot bath. The experiments 25 and 26 and 27 and 28 were also parallel experiments, bearing on both the questions we are considering.

TABLE I.

No.	Animal.	Time kept under water.	Put into hot bath at 100°. and how long.	Period after submersion at which thorax was opened.	Period after submersion up to which the ventricular portion of the heart continued to contract.
1	A rabbit	4 minutes	No	5th minute	11th minute, removed whilst contracting.
2	,,	6 ,,	No	7th ,,	25th minute.
3	,,	12 ,,	No	13th ,,	35th ,,
4	,,	20 ,,	No	21st ,,	26th ,,
5	,,	31 ,,	No	32nd ,,	Had ceased to beat when opened.
6	A large dog	14 ,,	No	17th ,,	Ditto.
7	A rabbit	11 ,,	No	12th ,,	16th minute.
8	A dog	8 ,,	No	9th ,,	34th ,,
9	A rabbit	5 ,,	Yes; 5 minutes	11th ,,	20th ,,
10	,,	5 ,,	No	12th ,,	Had ceased to beat.
11	,,	6 ,,	Yes; 4 minutes	11th ,,	30th minute.
12	,,	6 ,,	No	13th ,,	30th ,,
13	,,	8 ,,	Yes; 5 minutes	14th ,,	Had ceased to beat.
14	,,	10 ,,	Yes; 5 minutes	60th ,,	Ditto.
15	,,	10 ,,	No	60th ,,	Ditto.
16	A dog	8 ,,	Yes; 5 minutes	15th ,,	Ditto.
17	A rabbit	6½ ,,	Yes; 5 minutes	12th ,,	18th minute.
18	,,	6½ ,,	No	13th ,,	Had ceased to beat.
19	A dog	8 ,,	No	9th ,,	Ditto.
20	A rabbit	4 ,,	Yes; 4 minutes	9th ,,	Heart contracting irregularly when opened.
21	,,	4 ,,	No	10th ,,	Heart contracting regularly, 23 to the minute.
22	,,	4 ,,	Yes; 4 minutes	9th ,,	Ditto feebly.
23	,,	4 ,,	No	5th ,,	Ditto vigorously, 64 to the minute.
24	,,	2½ ,,	Yes; 3 minutes	6th ,,	No regular action, ceased at 10th min.
25	A cat	4 ,,	Yes; 4 minutes	13th ,,	Had ceased to beat.
26	,,	4 ,,	No	13th ,,	Heart beating regularly, continued up to 15th minute.
27	A rabbit	1½ ,,	Dipped in	10th ,,	Heart contracted up to 45th minute.
28	,,	1½ ,,	Yes; 2½ minutes	8th ,,	Ditto 25th minute.

In analysing the preceding experiments with reference to the first subject of inquiry, viz., the duration of the heart's action, two must be set aside, inasmuch as in them the thorax was not opened till after the lapse of an hour from the period of submersion; these are Nos. 14 and 15. Out of the twenty-six remaining cases, in eighteen the heart was found beating when first observed, in eight its action had ceased.

In taking *an average* of the period during which the heart continued to contract in these cases, Nos. 1, 20, 21, 22, and 23 must be omitted, for in them the organ was either removed whilst in action, or the time at which it ceased to beat was not accurately noted; the remaining thirteen give an average of twenty-five minutes *during which the ventricular portion of the heart continued to contract.* If we allow an average of nine minutes to the eight cases in which, on examination, the heart was found not contracting, and, including No. 1, at eleven minutes, take a general average of the twenty-two, we find it, as near as possible, *nineteen minutes.* These results do not coincide with those of Mr. Erichsen, who experimented on dogs by producing asphyxia in the open air; he gives an average of nine minutes and a half.

Setting aside the question of general average, it will be thus seen that, in eighteen animals out of twenty-eight, the heart was found contracting with more or less vigour and regularity when examined at the following intervals after the commencement of asphyxia, viz,, the 5th, 7th, 13th, 21st, 12th, 9th, 11th, 11th, 13th, 12th, 9th, 10th, 9th, 9th, 6th, 13th, 10th, 8th minute, and that it continued in action during periods varying from the 11th, up to the 45th minute from submersion.

In considering the second question; we have to examine it in two different ways. We have, first, to ascertain what are the pathological effects produced by the hot bath in an animal, in which asphyxia has been so long continued that, on the cause of the asphyxia being removed, no efforts at respiration are made. To do this we proceed to compare the morbid appearances presented by the various animals referred to in table No. 1, in order to see whether any differences exist between those which were submitted to the action of the hot bath, and those which were not so treated.

The chief morbid appearances observed in the animals which were not put into the bath were as follows :

The lungs were but little altered from their normal colour; their substance was less crepitant than natural, and their large, emergent vessels, as seen at their roots, were somewhat distended, but to no great extent. The right cavities of the heart and the pulmonary artery were much distended; the left cavities contained a small quantity of blood; the blood was black and fluid throughout the body, but coagulated when removed from the vessels; the arteries contained a small quantity of blood. On opening a pulmonary vein, or one of the large systemic veins, a considerable flow of blood always took place. It was observed during the examination of the animals that, whilst the heart was beating, and even after it had ceased to beat, on dividing a pulmonary vein, blood flowed, and continued to flow for a long time, and that the distension of the cavities of the right side of the heart, and of the pulmonary artery became much diminished, the blood flowing freely through the vessels of the lungs.

The general morbid appearances presented by the animals which were put into the hot bath were these. The lungs were much more discoloured than when the bath was not used; their large vessels were more distended; their substance was firmer and heavier. The cavities of the heart were loaded with blood, both those on the left side as well as those on the right. In some instances the blood was coagulated in the vessels of the lungs, in the systemic veins, and in the cavities of the heart, and in all there was more or less of this condition observed. When a vein was opened, the flow of blood was sometimes arrested by the presence of coagula, and was rarely free. The blood, when removed from the vessels, coagulated more rapidly, and formed a firmer clot than in the animals not put into the bath. In the animal which was put into the hot bath, after being drowned, for three and a quarter minutes, in water at thirty-six degrees, the morbid appearances were the same as in the other animals, except that there was even more congestion of the lungs. (This experiment is not referred to in the tables.) That there was actually, and not only apparently, increased congestion of the lungs, and an increased quantity of blood in them, is proved by the results of experiments Nos. 22 and 23, and 25 and 26, in which the weight of the lungs in both animals of each experiment was taken. The lungs of No. 23, the larger rabbit and the one not put into the hot bath, weighed *considerably less* than those of No. 22, the smaller rabbit and the one which was put into the hot bath; and, again, the lungs of No. 26, the larger cat, not put into the hot bath, weighed *less* than those of No. 55, the smaller cat, which was put into the hot bath.

The following are the respective weights of the lungs of the four animals:

	oz.	drs.	grs.
No. 22—The smaller rabbit (put into hot bath) .	0	4	49
„ 23.—The larger „ (not ditto) .	0	3	2
„ 25.—The smaller cat (put into hot bath) .	2	6	36
„ 26·-The larger „ (not ditto) .	2	5	37

The following notes are made with reference to the effects of the hot bath on the animals referred to in table No. 1:

In no single instance did the hot bath produce a respiratory effort, or any movement whatever, on the part of the animal. This was not only noticed in animals which had been drowned four, five, or six minutes, but in others which were under water only a minute, or a minute and a quarter, and which, on being taken out, did not at once begin to breathe; in no instance of this kind did the bath produce any satisfactory result. The contractions of the heart seemed, generally speaking, to be less vigorous, and to last for a shorter time, in those animals which were put into the hot bath than in the others.

In order to ascertain the second point referred to, viz., the effects produced by the hot bath on asphyxiated animals in which respiration has been re-excited, but is being only imperfectly carried on, a series of experiments was performed, which are referred to in the table No. 2.

These experiments were designed, as far as possible, to imitate, in the animals experimented on, the condition of an individual, who, after having been drowned, is recovered from the water, and on being exposed to the air, first of all gasps and then begins to respire feebly; they were also designed to test the value of the hot bath as a remedial agent in such cases.

The animals which were the subjects of these experiments were all rabbits, with the exception of one, which was a cat. The rabbits were all of nearly the same age and size. All the animals were drowned in water at the ordinary temperature, which varied from 45° to 50° Fahr.; they were kept under water, some for *one minute*, some for *one minute and a quarter*, and one, the cat, for *two minutes*. When removed from the water they were placed on a table exposed to the air, they were laid on their side, their mouth was opened, the tongue drawn forwards, and they were blown upon a few times by the mouth. In all the cases referred to, the animals began to gasp almost immediately they were laid on the table, and respiration soon became feebly re-established. In order to compare the effects produced by the hot bath with those where all treatment was omitted, except that of exposing the body to the open air, some of the animals were left to themselves, whilst others, as soon as respiration was feebly re-established, were put into the hot bath.

Altogether, thirteen experiments bearing on this part of the subject were performed, twelve being on rabbits, and one on a cat.

Of the thirteen, seven were treated by the hot bath, in which they were kept for periods varying from three and a half, to fifteen minutes. Of these, six died and one recovered.

TABLE II.

No.	Animal.	Time kept under water.	Put into hot bath at 100°, and how long.	Result.
29	A rabbit	1 minute	No	Recovered.
30	"	1 "	Yes; 3½ minutes	Died 5½ hours after submersion.
31	"	1 "	Yes; 15 minutes	Died 4 hours after submersion.
32	"	1 "	No	Died within 20 hours after submersion.
33	"	1 "	No	Died within 20 hours after submersion.
34	"	1 "	Yes; 7½ minutes	Recovered.
35	"	1 "	Yes; 12 minutes	Died 2 hours after submersion.
36	"	1 "	No	Recovered.
37	"	1 "	Yes; 10 minutes	Died 2 hours after submersion.
38	"	1¼ "	Yes; 5 minutes	Died within 20 hours after submersion.
39	"	1¼ "	No	Recovered.
40	"	1¼ "	Yes; 10 minutes	Died within 20 hours after submersion.
41	A cat	2 "	No	Recovered.

Six animals, after being removed from the water, were simply left to themselves, exposed to the atmosphere. Of these, four recovered and two died.

The following are the morbid appearances presented by the animals which died after having been put into the hot bath:

The lungs were generally small, as if collapsed; they were dark in colour, full of blood, quite firm, and almost liver-like in appearance; they resembled somewhat fœtal lung-tissue, or the collapsed lung-substance met with in adults. In some cases the condensed portions of the lungs sank in water. The trachea and bronchial tubes were free from frothy fluid; blood was found in all the cavities of the heart, the two ventricles bring generally about equally distended; the blood was never very fluid, and in one instance it was found coagulated in the cavities of the heart.

The post-mortem appearances of the two animals which were not put into the hot bath, but which died from the after-effects of the drowning, were as follows:—The lungs were somewhat congested, firm, and collapsed, but by no means to so great an extent as in the animals put into the bath. The blood was fluid, but less so than in acute asphyxia.

Without referring in detail to all the experiments, it appears to me that some of them show in a marked manner the different results of the two kinds of treatment resorted to; I am induced to quote the following:

In experiments 36, 39, and 40, the same animal, a young rabbit, was used. On the first occasion, it was drowned for *one minute*, and then left to itself on the table. It recovered well. Three days were allowed to elapse, during which the animal was quite lively, took food, and appeared, in no respects, to have suffered from the effects of the experiment. It was again drowned, this time for *one minute and a quarter*, and when removed from the water was again left to itself. It soon recovered, and next day was apparently quite well. Three days were allowed to elapse again, during which time the animal was quite lively, and ate as usual; in fact, no difference whatever was observable in its state as compared with that it was in before the first experiment. The animal was drowned for the third time, and for *one minute and a quarter*, as on the second occasion. When removed from the water it soon began to breathe. After lying on the table for about two minutes it was put into the hot bath; the breathing continued whilst in the bath, and the animal seemed to improve. It was kept in for ten minutes, and when taken out was able to walk. It seemed to go on tolerably well at first, but, five hours after submersion it appeared very sickly, and the respiration was hurried. Seven hours after the experiment the animal was alive; before the next morning, viz., within twenty hours of submersion, it was dead.

Although the experiments alluded to in the tables are not very numerous, yet, from the marked results obtained, it appears to me that they are sufficient to establish the principle of the injurious effects of the hot bath, at any rate on the class of animals which were the subjects of experiment; and I think we are justified in extending the principle to man. Many physiologists have deprecated the employment of the hot bath in asphyxia, but I am not aware that any experiments have ever been performed, previous to my own, which show its pathological effects on the lungs, and the manner in which, if had recourse to when respiration is feeble, it tends to destroy life some hours after its use. The recognition of these pathological facts must surely influence our practice, and tend to place our treatment of asphyxia on a more rational and scientific basis.

The following appears to me to be the way in which the hot bath acts:

By its stimulating influence it produces for a time an increased action of the heart, whereby more blood is sent to the lungs. Respiration being *nil*, the blood does not pass freely through the lungs, and hence their vessels become loaded. Some blood, however, finds its way to the left ventricle, which, from its imperfect action, is unable to get rid of its contents; the ventricle thus becomes distended, and subsequently arrested in its contractions. The circulation, although momentarily excited, ceases at an earlier period than when the bath is not used. Again, when respiration is being imperfectly carried on, the bath produces pathological results similar in their nature to those which it causes when respiration is suspended, viz., "engorgement" of the lungs, and subsequent arrest of the heart's action. In one instance, I have seen the hot bath produce convulsions, probably from causing an increased formation of carbonic acid, and its circulation in larger quantities to the nervous centres.

From the foregoing experiments and observations the following conclusions are drawn:

1. In dogs, rabbits, and cats, when asphyxiated by submersion, the ventricles of the heart do not, as a rule, cease to contract "in a few minutes after the cessation of the functions of animal life," but, in many instances, their action continues for a very considerable period; in all probability the same remark applies to man, and serves to explain how recovery has taken place after lengthened submersion, a circumstance which has been by many attributed to the fact of syncope having occurred at the period of immersion.

2. In cases of asphyxia where respiration has altogether stopped, the effects of the hot bath, are, to produce an accumulation of blood in the lungs and on the left side of the heart, together with a tendency to coagulate on the part of the blood. It does not tend to prolong the action of the heart, but rather to paralyze its movements and diminish

the duration of its contractions. It does not excite respiratory efforts, and prevents artificial respiration from being properly carried out.

3. In cases of asphyxia where respiration has been re-excited, and is being feebly carried on, the hot bath, although in some instances it seems to have no immediately bad result, yet has a tendency to produce a fatal issue, some hours after its use, by causing extreme congestion of the lungs, together with consolidation and collapse of the pulmonary tissue.

The practical inferences to be drawn from the above conclusions with reference to the treatment of asphyxia seem to me to be the following:

1. That efforts should be made to restore suspended animation in all cases where asphyxia has not been of *very* prolonged duration.

2. That, considering the effects of the hot bath, its prolonged use, whether respiration has ceased or is going on feebly, is not only inefficacious, but dangerous; and even to its temporary use—as, for instance, when the body is plunged into the bath and immediately withdrawn—there are serious objections, as it causes a loss of valuable time, and produces no direct benefit.

The object of the hot bath can only be to excite respiratory movements; these will probably be better brought about, so far as an influence of this kind can have any result, by alternately dashing hot and cold water over the body, or by the application of hot and cold cloths, etc.

3. That, judging from the increased mortality of the animals experimented on, which were put into the hot bath, as compared with those not so treated, it is safer practice, and more likely to lead to a favourable issue, to

omit all artificial aid in cases where respiration is going on feebly, than to make use of the hot bath.

4. That in the treatment of asphyxia all our efforts should be primarily directed to restoring, or continuing, as the case may be, the respiratory movements, and that all measures which have a tendency to load the lungs or embarrass the respiration should be avoided.

From my own observations in a case in which I succeeded in restoring suspended animation, from experiments I have performed on the dead body, and from the numerous instances of recovery that have taken place from its use, I am induced to believe that the best method we are yet acquainted with, of producing artificial respiration, is that recommended by Dr. Marshall Hall.

Although not directly bearing on either of the questions to which my experiments more particularly refer, there is one point in connection with the morbid condition of the lungs of the animals to which I wish to direct attention, viz., that whenever a pulmonary vein was cut, especially in those animals which were not put into the hot bath, the blood flowed freely, and relieved the distension of the right side of the heart and of the pulmonary artery. This fact seems to me to prove that, whatever may be the proximate cause of the arrest of the circulation in asphyxia, no condition, either of blood-vessels or blood, is produced, such as to cause a mechanical impediment to the passage of the latter fluid through the various vessels of the lungs; and the fact appears to me to oppose a strong argument against any theory, which would account for the cessation of the circulation, on the supposition that, either in the pulmonary capillaries or in the radicles of the pulmonary veins, such actual obstruction exists.

[Since the foregoing paper was read at the Royal Medical and Chirurgical Society, a committee of that Society have investigated the subject of apnœa, and they have recommended the use of Dr. Silvester's method, in preference to that of Dr. Marshall Hall, for performing artificial respiration. Undoubtedly, it appears, from the experiments which were performed by the committee, that a larger quantity of air can be drawn into the lungs by the former, than by the latter method; but it must be borne in mind that, in the treatment of the drowned, it is of the utmost importance to get rid of the fluid which is accumulated about the back of the mouth, and in the air-tubes. This cannot be accomplished by the Silvester method, as the body is kept in the supine posture. This circumstance appears to me to offer a serious, if not a fatal objection, to the use of the latter method in the first instance, in cases of drowning, although the objection does not apply to cases of suspended animation from other causes. Further, in the supine posture there must be a good deal of difficulty in keeping the tongue from falling back, and closing up the entrance into the wind-pipe. On the other hand, in the prone posture,—an essential part of the Marshall Hall plan,—this latter difficulty does not exist, and there is a means of escape provided for all accumulated fluids. It is not sufficient, in cases of drowning, simply to place the body in the prone posture for a few moments, preparatory to commencing the Silvester method, for all the frothy fluid present in the bronchial tubes cannot escape at once. I am, therefore, still of opinion, that in attempting to restore suspended animation from drowning, the Marshall Hall plan is to be preferred, and that it should not be relinquished, as long as there is an escape of fluids from the mouth; when this has ceased, the Silvester method may be used.]

CHAPTER XX.

OBSERVATIONS ON THE RHYTHMICAL ACTION OF THE HEART, AND ITS DURATION IN APNŒA.*

THE rhythmical action of the heart consists in the alternate contraction of the auricles and ventricles. This action commences at an early period of embryonic existence, and only terminates with life itself. The question as to the causes on which this rhythmical action depends has given rise to much discussion. The most generally received doctrine is, that it is due to nervous influence emanating from the ganglia found in the substance of the heart. The length of time during which the contractions of the heart continue in apnœa has been a matter of much inquiry amongst physiologists. The question is one of very considerable importance in connection with the treatment of suspended animation.

In a paper I recently communicated to the Royal Medical and Chirurgical Society of London, I expressed an opinion that the duration of the heart's contraction—*i. e.*, of its ventricular portion—was longer than had been generally supposed; and I stated that I had found the average time during which such contraction continued before it entirely ceased, to be—in the series of experiments I had performed on some of the lower mammalia—about nineteen minutes; in some cases the contraction lasting considerably longer. This statement refers, not to the rhythmical contraction of both auricles and both ventricles, but to the ventricular action alone.

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* This paper was originally published in the "Lancet" of July 20th, 1861.

In examining the question of the duration of the heart's beat, it is important that we should consider it in three points of view:—

1st. As to the time during which the rhythm of the whole heart lasts.

2ndly. As to the time during which the ventricular portion contracts.

3rdly. As to the order in which the different cavities cease to act.

With regard to the first point, the following experiments, recently performed, and which were accurately observed, may be referred to:—

Experiment 1. A healthy rabbit, nearly half grown, was drowned for five minutes, and, on being removed from the water, was at once opened. About a minute elapsed before the heart was seen. This brings us to the seventh minute of apnœa.

Observation.—The two auricles and ventricles were contracting rhythmically. The action of the left auricle was feeble, but that of the right cavities and of the left ventricle was strong, and well marked. The movements of the heart were watched, without the pericardium being disturbed, for several minutes, during which the rhythmical action continued. At the fifteenth minute of apnœa there was no perceptible contraction of the left auricle, but the right cavities contracted regularly; the ventricle after the auricle, and the left ventricle simultaneously with the right.

Experiment 2. A healthy rabbit, about three parts grown, was drowned for four minutes and three-quarters, and then opened.

Observation.—The heart was first observed about the middle of the sixth minute of apnœa. Both auricles and both ventricles were acting rhythmically. The contraction of the left auricle was feeble, but decided. Tenth minute: All the cavities were acting rhythmically; the action of the left side was feeble, that of the right stronger. At the fifteenth minute there was no perceptible movement of the left auricle; the other cavities were contracting. Sixteenth minute: The inferior vena cava and the abdominal aorta were divided; blood flowed from both vessels. Eighteenth minute: The left auricle was contracting again, and the whole heart acting rhythmically, and with more power than before the bleeding. Twenty-second minute: The heart was acting rhythmically, and regularly, thirty-four times in a minute. At the thirtieth minute, the two auricles were acting in concert, the ventricles responding, but not regularly. Observation ceased.

Experiment 3. A full grown cat was drowned for five minutes, and opened as soon as possible, viz., about the end of the seventh minute.

Observation.—Both auricles and both ventricles were acting feebly. There were two auricular contractions to one ventricular; the ventricular contraction immediately followed the second auricular. This continued for a short time, but at the twelfth minute there was but one auricular contraction to each ventricular action; in fact, the heart was acting feebly, but regularly, and in perfect rhythm. This lasted up to the fifteenth minute.

These experiments tend to show the duration of the rhythmical action of the heart in some cases. In the cat, it is true that when observed at the end of the seventh minute the rhythm was slightly disturbed, but it was subsequently restored, and it is therefore fair to assume that it might also have been restored, and the circulation re-established, if artificial respiration had been resorted to.

With regard to the second point, the experiments above detailed, and those to which I have referred in my communication to the Royal Medical and Chirurgical Society, are sufficient to show that the *ventricular* portion of the heart often contracts for a considerable time in apnœa. I have frequently seen very strong ventricular contractions in rabbits up to the twelfth, fifteenth, and twentieth minute.

It may be thought that the influence of the air on the surface of the heart may in some cases have tended to prolong its action. As my observations were made without the pericardium being disturbed, it is doubtful whether this could have had any such effect. But further, I have seen the ventricles in action when the chest was not opened until the periods I have mentioned.

With regard to the third point, I believe it will be found that the following is generally the order in which the different cavities cease to act; but as my observations on this point have not been very numerous, I do not insist on my conclusions. The left auricle usually ceases to act first; the right auricle and the two ventricles continuing to contract. Then the left ventricle ceases; subsequently, the right ventricle is arrested; and lastly, the right auricle, which often goes on contracting for a long time.

The great practical point in connection with this subject, as far as the treatment of apnœa is concerned, is—*Whether artificial respiration is capable of restoring the circulation:*

1st. When the action of both ventricles of the heart has ceased.

2ndly. When the right cavities alone are acting.

3rdly. When the left auricle has ceased to act, but the other cavities are contracting.

4thly. When the whole heart is acting rhythmically, but feebly.

1st. We have no proof that artificial respiration or any other means is capable of restoring the circulation when once the action of the ventricles has entirely ceased. Mr. Erichsen and Dr. Sharpey performed many experiments with the view of ascertaining this point, and the results always were, that although they could re-excite the circulation through the lungs, and sometimes reproduce the action of both auricles, they could never succeed in re-exciting the ventricles. "But, on the other hand, there is never any difficulty in re-exciting the action of this organ (the heart) if regular contractions of the ventricles are still continuing, however feebly or slowly."*

2ndly. I am not aware that we have any proof that when the right cavities of the heart alone are acting, the circulation can be restored by artificial respiration. My own belief is that such a result cannot be brought about.

3rdly. Can artificial respiration restore the circulation, supposing the left auricle alone to have ceased acting? The question almost resolves itself into this:—Can we re-excite the action of the left auricle when it has once ceased? Mr. Erichsen's and Dr. Sharpey's experiments distinctly prove that this can be effected. One of the experiments I have above detailed also shows this, for the abstraction of blood was followed by reaction of the left auricle. My belief is, that as long as the ventricles are acting in concert, and in rhythm with either one auricle or both, it is quite possible that a persistent use in artificial respiration may reanimate an asphyxiated animal; of course, the longer the use of such means is delayed the smaller will be the probabilities of success.

4thly. If it be possible to re-excite the circulation, even when the left auricle has ceased, *à fortiori* may we assume that this may be accomplished as long as the regular rhythm of the heart is in nowise disturbed, except as regards its frequency and power. It is during the continuance of this rhythmical action that efforts to reanimate are most likely to be successful; every moment of delay adds to the difficulty, and increases the danger.

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* Mr. Erichsen on Asphyxia. "Edinburgh Medical and Surgical Journal," vol. lxiii.

In a letter, which has been addressed by Sir B. C. Brodie (the late) to the Royal Medical and Chirurgical Society, referring to my paper on Asphyxia, that distinguished physiologist and surgeon states that he has never seen the rhythmical contractions of the heart continue after four minutes and a half of complete submersion. It is very possible that differences may exist in different species of warm-blooded animals as to the time rhythmical actions last, as well as in the individuals of each species. My observations have been chiefly made on rabbits, but also on dogs and cats. I have found that the duration of the heart's contraction in apnœa was longer in the first-mentioned animals than in the two latter.

It has been the opinion of many physiologists, that, in cases where animation has been restored in man after prolonged submersion, syncope has taken place at the period of immersion. To this point I have briefly alluded in my paper already referred to. That syncope occasionally takes place in drowning is highly probable; but of the fact itself it is obvious we can have no proof. It appears to me, however, that it is unnecessary to resort to this supposition to explain the recoveries which have taken place. From our knowledge of the prolonged action of the heart in the lower animals, and from facts, which we are acquainted with, in regard to man, I am induced to believe that, as a rule, there is, in the latter, a feeble action of the heart continued in apnœa for several minutes—an action which may be increased, provided the function of the lungs can be restored.

In considering the question of the duration of the heart's beat in apnœa, and the possibility of restoring animation, it is very desirable that, if we err, we should err on the right side. It is better that we should make *fifty* ineffectual attempts to save life, acting on the supposition of the prolonged duration of the heart's beat, than that we should suffer *one* life to be lost by allowing the opposite assumption to paralyze our efforts. That animation can be restored by artifical respiration, when normal respiration has ceased, is incontestable, and the exact time, beyond which no efforts can be of any avail, is by no means settled.

CHAPTER XXI.

THE APNŒA OF STILL-BORN CHILDREN.

A FUNDAMENTAL difference exists between the condition of the lungs of the still-born, and of the lungs of those in whom respiration has taken place; and an important distinction must be drawn between the apnœa of the former, and that of the latter — a distinction which involves a material difference of treatment. I am not aware that the point has been practically insisted on; and the same measures for effecting the entrance of air into the lungs of an infant who has never breathed, have been, and still are, recommended, as in the case of an adult.

I have advocated the use of Marshall Hall's ready method, in cases of suspended animation, when we meet with it after respiration has been fully established; and from the numerous instances of recovery which have been recorded, in still-born children, where this same method has been used, its applicability to such cases may appear to be well established.

A little consideration, however, will, I think, serve to show us that, in these latter cases, the method can be of no avail as a direct means of getting air into the lungs. The great feature on which the value of the ready method rests is this, *that by first compressing the chest, we drive air out of the lungs, and thereby bring into play the elasticity of the thoracic walls, by which means air is drawn into the lungs.*

By alternate compression and relaxation, a constant current of air to, and from, the lungs may be produced; but the *first* step in the process is the expulsion of air from the lungs. Now, in the still-born infant's lungs there is *no* air; the air-sacs, the bronchial tubes, and the trachea are unexpanded: in fact, they are in a perfectly solid state. It is quite obvious, therefore, that no air can be forced out of the lungs, and, consequently, that the primary step in the ready method cannot be accomplished. It is true, that if respiration have been established, however feebly, the method may be brought into play with benefit, but respiration cannot be commenced by this process.

The case is, however, far different, in all instances where the lungs have been previously in action. Here the essential condition for the performance of the ready method exists. Air is present in the lungs.

It appears to me, therefore, that the apnœa of the still-born child and that of the adult differ very materially; and in their treatment, although the same principles may be applicable, they must be modified to meet the requirements of each case. In the infant, respiration has to be primarily excited; in the adult, it has to be restored. In the infant, setting aside the question of direct inflation of the lungs,—a proceeding of very doubtful value,—all efforts should be directed to produce such an impression on the cutaneous nerves as will result in a reflex act of inspiration. In fact, we have to imitate the process of nature; the first inspiration is the result of the impression of air on the surface of the body. If this fail, the child is still-born, and impressions of a more powerful kind must be resorted to. The alternate use of hot and cold water to the skin, the brisk application of the palm of the hand to the back, friction of the chest, even rolling the body, may produce the desired result, a reflex inspiratory act.

And does not experience confirm these remarks? I have frequently been told, by those who have had to resuscitate still-born children, that the ready method has appeared to them of little, or no use; whereas means calculated to excite reflex action have been most efficacious. It is well that whatever plan we adopt, we should have distinctly before us the objects at which we aim. Those who are familiar with the condition of the infant's lungs before respiration has occurred, must, I think, feel convinced that, setting aside direct inflation, nothing can be of any avail to get air into them, except the expansion of the chest-walls by muscular action.

PART II.

DISEASES OF THE HEART, AND THORACIC ANEURISM.

PART THE SECOND.

DISEASES OF THE HEART, AND THORACIC ANEURISM.

DISEASES OF THE HEART.

CHAPTER I.

ON THE SOUNDS OF THE HEART.

(LECTURE.)

GENTLEMEN,—In the present lecture, I propose to consider the causes of the sounds of the heart. The subject is one on which the minds of physiologists seem to be by no means settled; and a brief inquiry into the different causes which have been, or still are, assigned for the production of the sounds, will, I venture to hope, not be unattended with benefit.

A knowledge of the action of the heart, and of the causes of its sounds, is of great importance to you in the diagnosis and treatment of cardiac diseases. If you do not master these elementary points, you will never be able to appreciate the changes which disease produces; whereas, on the other hand, if you make yourselves thoroughly acquainted with them, a little practice in the wards of your hospitals will enable you to recognize, and understand the morbid sounds, or murmurs, which are so frequently met with.

Our improved knowledge of cardiac diseases affords, I think, a striking instance of the value of physiological observation. To the investigation of the movements of the heart, of the causes of its sounds, and of the nature of its rhythm, no little labour has been devoted during the past forty years; and although on some points, connected with these phenomena, differences of opinion still exist, yet, on the more important points, those especially which bear on pathology and diagnosis, it may be almost said that unanimity prevails.

Before I speak to you of the sounds of the heart, let me say a few words on another subject, on which great diversity of opinion has existed, viz., the manner in which the auriculo-ventricular valves are closed. That the *musculi papillares* aid in the closure of these valves, by drawing the flaps from the sides of the ventricles, during the ventricular systole, has been the opinion of many physiologists; but experimental observation has shown that the only function these muscles perform, is, to support the valves against the column of blood, and to prevent them from being pushed back into the auricles — in fact, to hold the valves in such a manner that they may form a resisting roof to each ventricular cavity.

From experiments made by pouring fluid into a recent heart, stripped of its auricles, the following seems to be the manner in which the valves close.

When liquid is poured into one of the ventricles through the auriculo-ventricular opening, the flaps of the auriculo-ventricular valve rise on the surface of the liquid, and when the ventricle has become fully distended, as it is just before it is about to contract, the flaps have risen, so as to form a perfect septum between the auricle and ventricle. In order to account for this action on the part of the valves, their rising on the surface of a fluid lighter than themselves, and against the direction of the current passing through the orifice which they arm, it has been recently shown, by Dr. Markham, that they possess, in their structure, a quantity of yellow elastic fibres, which curl up, and thus draw the margins of the valve upwards, and inwards, towards the centre of the opening. The knowledge of the existence of these fibres renders more easy the explanation of the peculiar mode of action of these valves, and affords another proof of the importance of minute anatomical enquiry in all that relates to physiological action.

But to pass on to the sounds of the heart. If you place your ear over the præcordial region, you will hear two sounds: one, heard most distinctly towards the apex of the heart, dull and somewhat prolonged; the other, heard most distinctly at the base of the heart, less prolonged and sharper in its character. These sounds are called, respectively, the first sound, and the second sound, of the heart. The first is synchronous with the contraction of the ventricles and the propulsion of the blood into the aorta and pulmonary artery, and slightly precedes the pulse at the wrist. The second immediately succeeds the first, and is synchronous with the relaxation of the ventricles, and the attempt of the blood to regurgitate from the aorta and pulmonary artery.

If you consult the different works on physiology and medicine, where reference is made to the causes of these sounds, you will find that, although there is a more general agreement than there was some years back, yet, the views of authors are by no means settled on the subject. The difficulty seems to be with the first sound; for the experiments of Dr. Hope, performed many years ago, proved that the second sound was due to the closure of the two sets of semilunar valves.

Let us now examine into the events, which are taking place in the heart, at the time when these sounds are produced, and then, ascertain the opinions which are generally held, with reference to their causes, at the present day.

The events which correspond with the first sound are — first stage of dilatation of auricles; contraction of ventricles; closure of auriculo-ventricular valves; opening of ventriculo-arterial valves; propulsion of blood against auriculo-ventricular valves, and through the orifices of aorta and pulmonary artery; impulse of heart.

The events corresponding with the second sound are — relaxation of ventricles; dilatation of auricles; backward flow of blood in aorta and pulmonary artery towards ventricles; closure of semilunar valves.

From the time of Laennec up to the present day, as many as twenty-nine theories have been proposed to account for these sounds. It would occupy too much time to refer to all these theories, and I must content myself by drawing your attention to the principal ones which are held at the present day.

We have, as alleged causes—1. Impulse. 2. The rush of blood through the narrowed orifices of the great arterial trunks. 3. The collision of the particles of blood with one another, and with the parietes of the heart. 4. The opening of the semilunar valves. 5. Muscular sound,—bruit musculaire. 6. The closure of the auriculo-ventricular valves. Such are the theories with regard to the first sound. With regard to the second, scarcely any difference of opinion exists; it is acknowledged to be due to the closure of the semilunar valves.

Let us now examine the various causes above alluded to. That impulse has nothing whatever to do with the first sound, is most satisfactorily proved by the following experiment:—If the anterior wall of the thorax of one of the lower animals (a large dog or a donkey) be removed, and the stethoscope be applied to the surface of the heart itself, the sound is found in nowise diminished in intensity, but, if anything, it is rather more distinctly heard than before. This observation was made by Dr. Hope. It has been confirmed by all subsequent experimenters, and I have frequently had opportunities of verifying it myself. The impulse, in fact, does not partake of the nature of a blow against the chest walls; it is not produced by the apex of the heart impinging against them, but it results from the ventricles, in their contraction, assuming a globular form, and, consequently, pushing out the yielding intercostal spaces. When the heart of a living animal is grasped by the hand, the impulse or bulging of the muscle is felt on all sides, and not more at one part than at another; it is felt on the under surface of the diaphragm, when the posterior wall of the heart is touched through that muscle, as well as on the anterior surface, and at the apex of the ventricles. Further, that the impulse is not the result of a blow on any particular spot, is proved by the fact that it is by no means confined to one spot. It is, usually, in health, most distinct in the fifth intercostal space, but it may be frequently felt in the fourth; and, in cases where the heart is enlarged, the extent of surface over which the impulse becomes sensible is much increased. It is thought by some physicians that, although the impulse does not contribute to the intensity of the first sound, under ordinary circumstances, that it does so, in cases of increased action; and that the "rapping" noise, as it is called, of the nervously-excited heart, is the result of the impulse. This explanation of the exaggerated sound, under these peculiar conditions, does not appear to me satisfactory. The sudden, and violent closure of the valves is, I think, quite sufficient to account for the intensity of the sound.

I pass on now to consider the second assigned cause of the first sound, viz., the rush of blood through the narrowed orifices of the great arterial trunks. This view is assumed from the fact, that sound is produced by pumping fluid through tubes out of the body; and further, that, whenever the calibre of a vessel in the body, through which blood is circulating, is diminished, a sound is produced. That such analogy as is here assumed, exists between the forcible, and sudden pumping of fluid through an inert tube out of the body, and the action of the heart in impelling its blood into the elastic and yielding vessels situated at its base, I cannot admit; nor is the condition of these vessels at their origin at all similar to that of a compressed artery. There is no natural impediment to the onward flow of the blood; the parts leading to the vessels are perfectly smooth, and the vessels themselves are of so elastic, and yielding a material, that they dilate in every direction when they receive the blood, as can be readily felt if they are grasped during systolic action of the ventricles. If the arterial orifices were compressed during the time the blood is passing through them, so that their shape would be altered, and their calibre diminished, the same result would take place as in the arteries elsewhere; but no such compression occurs, nor is there any increase of sound over the base of the heart, as there ought to be, if this view were correct; on the contrary, when the heart is exposed, and a stethoscope is applied to it, the sound is found to be most intense over the situation of the auriculo-ventricular valves. To suppose that the passage of the blood from the ventricles to the arteries would be attended with sound, is to suppose that the blood meets with an impediment to its course, which is certainly not the case. Further, I would remark that, the ventricles being filled with blood before they contract, there is a gentle, and not a violent propulsion of it into the vessels, and that these, as they receive it, yield in proportion to the force with which they are distended — an additional reason for the non-production of sound. Were the blood propelled into cavities filled with air, or partly with air, and partly with blood, we should expect a sound to be produced; but what is the case with regard to the distension of the ventricles? Their walls are in contact at the end of each systole, relaxation of the fibres takes place, and blood is immediately poured in from the auricles by the *vis-à-tergo*. This, gradually and silently distends the cavities, and at length the auricular action brings them to the necessary condition to produce contraction. There is no element of sound here; there is no splashing of the fluid blood with a fluid gas; all the actions are going on in air-tight cavities. As well might we expect to produce a sound, by succussing a man with effusion in the pleura, as to expect that the current of blood in the chambers of the heart, in their normal state, would give rise to a sound; but once admit the air, another element is present, and sound is elicited.

But there is another view which has been advanced, in reference to the passage of the blood from the ventricles to the arteries, and of the mode in which sound is thereby produced. It has been maintained that the forcible impulsion of the ventricular blood, against the closed semilunar valves, with a column of blood above them, forms an important element in the first sound of the heart — that, in other words, the concussion of the active (ventricular), and passive (arterial) portions of the blood must give rise to sound.

Now, I may remark with reference to this view, — that concussion takes place between the passive and active columns of blood,—that the nature of the concussion is such, that we could scarcely expect it to produce a sound. During the time the ventricles are filling, the blood in them rises to the level of the under surface of the semilunar valves, and when the cavities contract the fluid pushes open the valves, and communicates its force to the column in the vessels. There is no propulsion of the blood against the passive column from a distance; there is no space between the blood which fills the ventricles, and that in the vessels; one column lies above the valves, the other beneath them. It is contrary to all we know of the phenomena which accompany the passage of liquids in closed tubes, that a sound should be produced under such circumstances.

Some physiologists have expressed an opinion, that the collision of the particles of the blood amongst each other, during the ventricular systole, contributes to the first sound. I think it is a sufficient answer to this view, that, during other periods of the heart's rhythm, such collision produces no sound. The blood is poured into the auricles; from auricles to ventricles — even forcibly, during the contraction of the former; it is, further, injected against the irregular walls of the ventricles, which present a condition, from the arrangement of the columnæ carneæ, which ought, in my opinion, to elicit a sound, if such collision were capable of producing one. But, notwithstanding this, these actions are silent. Are we, then, to conclude that what does not produce sound at one time produces it at another, without the existence of any essentially different condition?

The opening of the semi-lunar valves, the fourth assigned cause to which I have referred, has been considered by some physiologists to assist in the production of the first sound. I shall not dwell long on this supposed cause, as I consider it cannot in any wise assist in the production of the first sound. When we reflect on the condition of the valves at the moment of systole,—that they have a column of blood above, and a column of blood below them, that they are not forcibly thrown against the sides of an empty vessel, that the blood on the upper surface has to be displaced, and that this must prevent the shock of the valves against the walls of the vessel,— it is impossible, I think, to admit that any element of sound exists therein; and such is the view taken by most physiologists.

I pass on, now, to the consideration of the fifth assigned cause, muscular sound — bruit musculaire. That the muscular structure of the ventricles must necessarily produce a sound in its contraction, appears to have been generally admitted as an established truth, and no one, until somewhat recently, took the trouble to put the question to the test of actual experimental examination, in such a manner as to elicit, in my opinion, a safe and satisfactory reply.

If you place your ear, or a stethoscope over a muscle during its contraction, you will hear a sound, but, in its character, it differs entirely from the first sound of the heart. Perhaps you cannot have a better example of the sound produced by muscular contraction, than that afforded by the action of the masseter muscle. If, when your head is resting on a pillow, you forcibly contract your masseter muscle, you will hear a rumbling noise — a noise which continues during the contracted condition of the muscle, and is also heard during its relaxation. The exact cause of this sound I am unable to tell you. It may be due to the change in shape of the muscular fibres—it may be due to the alteration of the circulation— or to some other cause. Kiwisch is of opinion that the sound is produced by the vibration of the air in the meatus, and in the stethoscope; and he says that, if this source of error be avoided, no sound is heard. At all events, I think, the more you examine this sound, and all other muscular sounds, the more you will be convinced that they bear no resemblance to the first sound of the heart.

Before alluding to the experiment which, in my opinion, proves, beyond all doubt, that the contraction of the ventricles produces no sound, I think it right to bring under your notice, other arguments which tend to prove it. If the ventricles during contraction produced sound, it is but fair to infer that the auricles would also produce sound. We find, however, that this is not the case. If it be said that the muscular substance, of which the walls of these cavities are composed, is so slight, that no audible sound could be expected from its contraction, I maintain that such an argument is untenable; and I would ask, What is the exact amount of muscular fibre necessary to produce an audible sound? Are not the auricles of a full-grown man as large, and do they not contain as much muscular fibre, as the ventricles of a *fœtus in utero?*—and shall we admit that the contraction of the latter will produce the clear audible click, with which many of you are familiar, and deny that the former can have any such effect at all? Again, presuming that the auricular fibres in man are not sufficient in number to produce a sound, are there not animals which possess auricles larger than the ventricles of some adult men? but yet, if you listen to the sounds of the heart in these animals — as, for instance, the ox or the horse — you will find that they are the same in number and character as those of man; during the auricular contraction, no sound is heard. These facts seem to me conclusive against the muscular theory.

We are not, however, without positive evidence that the ventricular contraction produces no sound. To Dr. Halford, who, some years ago, brought the subject under the notice of the profession, we are indebted for the beautiful experiment to which I shall now draw your attention.

The experiment consists in depriving an animal (a dog or a donkey) of sensation by means of chloroform; and, whilst artificial respiration is kept up, the anterior part of the thorax, and the pericardium are removed, and the heart is exposed. But let me quote Dr. Halford's own words, he says:—

"My proceedings were as follow: Large dogs were obtained, and as in my preceding experiments (the animals being under the influence of chloroform), the heart was exposed, and the circulation kept up by artificial respiration. A stethoscope being applied to the organ, the sounds were distinctly heard. The superior and inferior venæ cavæ were now compressed with bull-dog forceps, and the pulmonary veins by the finger and thumb; the heart continuing its action, a stethoscope was again applied, and neither first nor second sound was heard. After a short space of time, the veins were allowed to pour their contents into both sides of the heart, and both sounds were instantly reproduced. The veins being again compressed, all sound was extinguished, notwithstanding that the heart contracted vigorously. Blood was let in, and both sounds were restored. I have thus frequently interrogated the same heart for upwards of an hour, and always with the like result."

Dr. Halford further observes, "All that is claimed for the above experiment, is its exemption from any rude interference with the mechanism of the heart's action. The cavities of the heart are untouched; there is no finger thrust into the auricle or ventricle; no hooking back of valves: in fact, not one source of sound substituted for another. Both sounds are destroyed and reproduced by the same means; the strongest argument for their both depending on the same cause, which is simply the backened current of blood, first against the auriculo-ventricular, and second against the ventriculo-arterial valves."

Now, what is the value of this experiment? Does it not unquestionably prove that there is no muscular sound in connection with the systole of the ventricles of the heart? and does it not disprove all theories of the cause of the first sound founded on a contrary supposition? But some objections have been raised to the conclusions which have been drawn from this experiment. It has been asserted that, in the experiment, the contraction of the ventricles is not like that of the ventricles when they are distended with blood; and that the experiment does not prove that the ordinary contraction produces no sound.

In answer to this, I would remark that the shortening of the muscular fibres takes place, the muscle assumes its globular form, and in fact is placed in that condition in which it ought to elicit a sound, if the muscular contraction could do so. Further, the so-called muscular sound (of voluntary muscles), which is said to resemble the first sound of the heart, takes place during the contraction of the fibres, without the muscle having any substance to contract upon. I am aware that some of those who have witnessed the experiment have expressed a doubt of the facts, and believe that they heard a sound. Now, as they, probably, only saw the experiment performed in a crowded theatre, where there must necessarily be much trouble in maintaining absolute silence, and much difficulty, during a hurried examination, in discriminating between a sound of rubbing against the stethoscope, and one produced in the muscle, their statement can scarcely be allowed to have any weight, in contra-distinction to that of many who have borne opposite testimony. I have often satisfied myself, by careful examination, of the truth of Dr. Halford's assertion. I assisted Dr. Halford in his original experiments, and also in those which he performed in our school, and I have, frequently, under circumstances to prevent all possible mistake — absolute silence, the presence of few attendants, etc. — convinced myself of the noiselessness of the ventricular contraction.

I have thus endeavoured to show you that, neither the impulse of the heart, the rush of blood through the orifices of the arterial trunks, the collision of the particles of blood against each other and against the walls of the ventricles, the opening of the semi-lunar valves, nor lastly, the contraction of the muscular fibres of the ventricles, contributes to the first sound of the heart.

Now, let me say a few words in reference to the remaining alleged cause, the closure of the auriculo-ventricular valves.

That the closure of the auriculo-ventricular valves is an element in the production of the first sound has been generally admitted; and having, as I believe, eliminated the other elements which I have put down as assigned causes, I hope I shall be able to convince you that the tension which these valves necessarily undergo, during the contraction of the ventricles, is attended with sonorous vibrations, on which the first sound solely depends. I believe that Dr. Billing, in 1832, was the first to place the valvular theory of the heart's sounds fairly before the profession; and, in the same year, Rouanet advanced and supported the theory, in a thesis. Both Billing and Rouanet worked independently, and the view may have been original with both. The theory found a warm supporter in Bouillaud (who, however, thought that the opening of the semi-lunar valves contributed to the sound), whose work on the heart contains an able analysis of the views current at the time it was written. The view was also ably advocated by Mr. Bryan, in the pages of the 'Lancet.'

Dr. Billing, writing in 1832, says:

"The first sound is caused by the tension produced in shutting the auriculo-ventricular valves, and the second sound is caused by the tension produced in the shutting of the ventriculo-arterial valves."

Again he writes, in 1833, in the 'Lancet':

"The first sound takes place exactly synchronous with the impulsion and action of the ventricle; hence it might be supposed that the action of the muscle (as averred by some) produces the first sound. But the second sound takes place when there is no action of the heart going forward; and this is peculiarly evident when there is an intermitting pulse, as there is then a marked pause after the second sound; so that, in fact, there is nothing but the semilunar valves in operation to produce sound at the instant."

Rouanet, in his thesis, says:

"Numerous experiments have proved to me that every membrane, on passing from the state of relaxation to that of sudden tension, yields a sound, which varies according to circumstances, and is more or less intense in proportion to the powers of tension. Its tone increases with the fineness and extensibility of the membranous tissue. The greater size, thickness, and extensibility of the membrane render the sound duller, and the substance to which it is attached modifies the quality of sound in proportion to its thickness, softness, and elasticity."

In applying these principles to his theory, M. Rouanet attributes the first sound to the sudden approximation, and tension of the mitral and tricuspid valves, during the ventricular systole; and the second, to the brisk tension of the sigmoid valves, owing to the counter shock of the column of blood in the aorta and pulmonary artery. He says:

"The first sound is loud, and depends to a certain extent on the energy of the ventricles, and is duller than the second. The valves which occasion it are large, and the walls which conduct it thicker. The second sound is sharper, because the valves are smaller, thinner, and attached to more sonorous walls."

Now, I believe that in these few observations of M. Rouanet, so simply and clearly expressed, the whole subject is contained, and that he has given a truthful explanation of the phenomena. It is unnecessary to quote the experiment he performed, in which he imitated the second sound of the heart, by allowing a column of fluid to fall on the sigmoid valves.

Working at the same subject, and unacquainted with the labours of Dr. Billing and M. Rouanet, Mr. Bryan, in 1833, published a series of papers of great value on the movements, and sounds of the heart. He advanced, and most ably defended, the valvular theory. Amongst other remarks, he has the following:

"Any flexible solid suddenly brought from a state of relaxation to a state of tension, vibrates, and the vibrations are sonorous or not, *i.e.*, audible or not, according to its physical structure.

"At the commencement of the systole of the ventricles, the auricular valves are flapped into play, and at the instant of their closure the whole substance of the ventricles, and the valves are suddenly brought to a state of tension, and then consequently vibrate. I leave it to the reader to determine, according to the laws of physics, whether the vibrations of the valves, floating freely in fluid, or the muscular substance of the ventricles, trammelled by the contact of surrounding solids, would most contribute to the formation of the first sound."

With a view of showing that the tension of the valves is capable of giving rise to a sound, Mr. Brakyn devised the following ingenious experiment. An ox's heart being procured, a bladder is connected with the left ventricle by means of a flanged tube; with the aorta is connected another bladder, and this latter has a tube passing from it, which is connected with a third bladder attached to the left auricle. The tube, between the second bladder and the third, has a metallic portion with a stop-cock in it. This arrangement of bladders and tubes allows of the passage of currents of air, representing a mimic circulation through the left heart. The experiment is performed as follows: the bladders being inflated, and the stop-cock closed, pressure is made on the auricular bladder, and the air is injected into the left ventricle, and ventricular bladder; the latter is compressed, and the air, forcibly impelled upwards, produces a closure of the auricular valve, and passes into the aorta, and the bladder connected with it; this bladder is now suddenly compressed, the sigmoid valves close, and the air is prevented passing back into the ventricle. The phenomena attending these actions are two sounds, one when the ventricular systole is imitated, and the auricular valve closed, and the other when the elastic reaction of the aorta is imitated, and the semilunar valves are closed.

In remarking on his experiment, Mr. Brakyn says:

"The sounds being produced without any muscular contraction, rush of blood, etc., must evidently be valvular . . . the first sound is as perfect as the second. . . . The illustration, though conducted through air, ought to be conclusive, inasmuch as a suddenly strained membrane which gives a tympanic sound in air, will do the same in water also, as I have tried, but not so loudly."

From having repeated the above experiment, I quite agree with the observations of Mr. Brakyn; the sounds resemble, in my opinion, those of the heart as closely as possible, bearing in mind the nature of the fluid in which they are produced. The first is a duller and more prolonged sound than the second, and differs very little from the corresponding clear sound sometimes heard in a dilated and thin heart.

Subsequently to the experiments of Mr. Brakyn, came those of Dr. Halford, which I have already detailed; and, although they do not directly prove the truth of the valvular theory, they afford positive evidence of the silent contraction of the muscle, and, in my opinion, leave no other cause for the sound than that of valvular tension. Putting together the experiments of Brakyn and Halford, it appears to me that experimental evidence of the theory we are examining is complete; the investigations of the former, taken alone, might be deemed inconclusive, as they afford no proof that muscular action does not assist in the sound; but on this latter point, the experiments of the latter are decisive.

With regard to the second sound, as I have already told you, there is scarcely any difference of opinion. The experiments of Hope proved that it was solely due to the closure of the semi-lunar valves. When the arteries recoil after being distended by the ventricular systole, the blood in them is forced back towards the ventricles. By this act the semi-lunar valves are brought into play, they are stretched across the vessels, and suddenly made tense, and thus sound is elicited.

If you listen to the sounds carefully, you will find that they differ in degree, merely, and not in kind. The auriculo-ventricular valves are large, thick, and strong; consequently, their vibrations are slow, and the sound they produce is prolonged. On the other hand, the ventriculo-arterial valves are small, thin, and comparatively weak, their vibrations are rapid, and the sound they produce is short. Both sounds may be illustrated by making tense two pieces of membrane of different size and thickness.

I hope I have said enough to convince you that the two sounds are simply and solely valvular; that the first results from *the tension of the mitral and tricuspid valves, produced* *by the blood being forcibly propelled against them,* and that the second is due to *the tension of the semi-lunar valves of the aorta and pulmonary artery, produced at the time of their closure.*

I have dwelt somewhat at length on this subject, because I attach great importance to it in connection with the diagnosis of cardiac diseases. If this simple theory of the sounds of the heart be true, it is well that it should be widely recognized, and it appears to me that the modifications of the sounds, which we hear in disease, tend in the strongest possible manner to confirm it. If you hear a murmur in listening to the heart, you know, at once, that there is something wrong with the valves, you do not think of the muscular walls; you know that there must be some deposit in connection with the valves, which, either from its roughness, causes a sound as the blood passes over it, or else prevents the valves from closing properly, and thus allows of regurgitation. Moreover, the alterations in the sounds which result from changes in the muscular walls of the heart afford, in my opinion, further evidence of the truth of the valvular theory; but I must not stop to discuss this point now.

CHAPTER II.

PERICARDITIS.

(CLINICAL LECTURE.)

GENTLEMEN,—I wish to call your attention, to-day, to the subject of pericarditis, and it is especially with reference to the treatment of the disease that my remarks will be directed; and the point to which I shall first refer is, the propriety, or impropriety of exhibiting mercury in the affection.

The value of mercury, in the treatment of acute pericarditis, has been for some years past a disputed point amongst practical physicians. At one time the drug was almost universally administered, wherever the symptoms of pericardial inflammation existed; subsequently, its power to control the disease became questioned; and at the present time, although we find some physicians advocating, and resorting to, its use, the majority of practitioners are fast losing confidence in its efficacy, and belief in the necessity for its exhibition.

Setting aside those cases of pericarditis which come on during the progress of Bright's disease, in which it is universally admitted that mercury ought not to be given, we find that, in the great majority of cases, the affection is coincident with rheumatic fever; that it is the result of the rheumatic condition of the blood, and must therefore be regarded as a local manifestation of a constitutional malady. In the treatment of acute rheumatism, when the joints alone are affected, we do not consider it necessary to administer mercury; we know that, without the exhibition of the drug, the inflammation will subside, the effused matters will become absorbed, and the joints will return to a healthy state.

In pericarditis, however, an organ is involved of far more importance than the joints; there is danger that the effused matters may be so abundant as to paralyze the movements of the heart, or that exhaustion may result from the severity of the local affection. It therefore becomes important to consider whether more active measures are not required, than when the rheumatism is simply articular.

Let us briefly consider the objects which we should have in view in the treatment of a case of pericarditis, and the possible results which may be achieved by treatment. When we recognize, from the presence of friction-sound, together, perhaps, with increased dulness on percussion, unmistakeable evidence of the disease, can we expect that we shall be able to promote the absorption of all the effused matters, so as to restore the pericardium to its normal condition as a serous membrane?

I believe that when pericarditis terminates in recovery, the most frequent way in which it terminates is by adhesion. I do not mean to deny the possibility of recovery taking place without any adhesion, but, in my own experience, I cannot say that I have ever met with such a case; and I have every reason to believe that, where recovery has followed well-marked symptoms of pericardial effusion, there has been more or less complete adhesion of the two pericardial surfaces. In slight cases of the disease, and where the pericardium is only partially affected, no doubt recovery takes place without any adhesion being produced; and we see the proof of this in *post-mortem* examinations. But where the disease has been either extensive or severe, I doubt the possibility of the pericardial membrane being restored to a healthy state, and

I believe that adhesions, more or less extensive, are always formed.

There are some cases, such as the following, which are occasionally met with, and which may give rise to the supposition, but I think without any just grounds, that effused fibrine has been completely absorbed.

Edward F., a Frenchman, aged 42 years, a ship-carpenter, was admitted into the Hospital, under my care, on the 27th of March, 1861. He had left New York about three weeks before admission; and, during the voyage, had been more or less ill with rheumatism. He, however, continued at his work up to five days before he came here, when he was so ill, that he was obliged to take to his bed.

When seen on the 28th of March, he complained of great pain in the legs and arms; the joints were swollen and tender, there was some anxiety of countenance, but he had no pain in the chest; the heart-sounds were normal, but faint. He was ordered a grain of opium three times a day, bicarbonate of potash, and a belladonna lotion to the joints.

On the 30th, he complained of pain in the left side on taking a deep inspiration. The pulse was small, 120; respirations, thoracic, 40 per minute. There was no pain on pressure over the cardiac, or epigastric region. *There was no increased dulness in the cardiac region. A loud, grating, double friction-sound was heard all over the front of the heart.* He was ordered a purgative, and the opium and potash were continued.

On the 31st there was less pain in the limbs; the pulse was 104, and stronger. *The pericardial friction-sound was not audible over any part of the heart, either when the stethoscope was lightly applied, or applied with pressure.* There was pleuritic friction-sound in the left axilla. Six leeches were ordered to be applied to the left side, and three grains of calomel and one of opium were given at bed-time.

He experienced great relief from the leeches; and on the following day (April 1st), the pulse was 102, quite regular; the respirations were 30. Friction-sound was still heard in the left axilla, but not in the precordial region.

On the 2nd of April the pulse was 82, and stronger; respirations, 36. He had passed a restless night, and had been slightly delirious. There was no pericardial friction-sound, nor endocardial murmur audible. An ounce of port wine was ordered every three hours, and the following mixture:—Solution of acetate of ammonia, two ounces; bicarbonate of potash, two drachms; tincture of opium, eighty minims; water to eight ounces: an ounce every three hours. For diet he took beef-tea, arrowroot, and milk.

On the 3rd he had passed a good night. Pulse 84; respirations 32. There had been slight delirium. On the 4th he had slept well, and there had been no appearance of delirium. Pulse, 82; respirations, 30; heart-sounds clear; pleuritic friction-sound still heard. On the 6th, the wine was diminished to six ounces; and on the 8th it was stopped. He subsequently had slight return of pain in the joints, for which he took iodide of potassium and Dover's powder.

He was discharged, quite well, on the 25th. The heart's action was regular, and there were no symptoms of any diseased condition of the organ whatever.

I believe that, in the above case, the friction-sound, which, it will be observed, was only heard on one day, was due, not to any fibrinous effusion, but to a dry condition of the pericardial membrane. The incipient inflammation subsided rapidly, and did not reach the stage of effusion. The friction-sound was well marked, and was heard by others as well as by myself; it was not accompanied by any signs of effusion, and there were no symptoms, either at the time or afterwards, which would justify the supposition that adhesion had taken place, in the twenty-four hours that elapsed between the first recognition of the sound, and its final cessation.

The occurrence of delirium in the progress of the case, and its subsidence under the administration of stimulants, is a point of interest. To the influence of stimulants, in relation to this particular symptom, I shall refer hereafter.

In a therapeutical point of view, a recognition of the mode in which pericarditis terminates is of great importance; for if our object is to promote adhesion as soon as possible, we ought to abstain from the use of remedies which, we know, have a tendency to diminish the plasticity of the blood, and to favour liquid effusion—such, for instance, as mercury.

No doubt, those who administer mercury in pericarditis can show a large per-centage of recoveries; but I think that an appeal to the statistics of the period, during which mercury has not formed a general element in the treatment of the affection, would show a smaller per-centage of deaths than in former years. From my own experience, I am strongly in favour of a non-mercurial mode of treatment; and for some years past I have never given mercury. The only fatal case of acute pericarditis, uncomplicated with Bright's disease, which I have had amongst my hospital cases during the past eight years, was one in which I gave mercury to salivation. In this case extensive effusion took place, which the mercury in nowise seemed to control.

The case was one in which there was but slight rheumatic affection of the limbs, and the pericarditis was preceded by an attack of pneumonia, from which the patient was recovering at the time the pericardial inflammation set in.

The following are the notes of the case:—

James S——, aged 35 years, a seaman, of fair muscular development and moderately stout, was admitted into the hospital on the 26th March, 1860. Five days before admission, he began to feel unwell, but continued his work up to the 24th. He had suffered from pain in the right side, and in the lower limbs, and had been hoarse for about two days. There had been no rigors; he had slept badly.

On admission, he complained of a troublesome, irritating cough, with pain in the right side. He was very hoarse. Pulse 100, moderately full; the skin was rather hot, but not pungently so; the tongue was foul, and the expectoration frothy. There was crepitation over the back of the right lung, with bronchophony and bronchial breathing; there was dulness on percussion as high as the spine of the scapula, and in the right axilla, with increased vocal resonance on the right side; there was deficient movement of the chest walls. On the left side the resonance on percussion and breath-sounds were normal.

He was ordered an eighth of a grain of antimony, with camphor mixture, every three hours; turpentine fomentations to the chest; and low diet.

March 28th.—He had slept a little, and felt easier, and free from pain. The pulse was 100; the skin rather hot; the sputa were scanty, adhesive, but not rust-coloured. There was loud tubular breathing and bronchophony opposite the lower part of the scapula; otherwise the auscultatory signs were unchanged.

March 29th.—The pulse was 100; the skin moist; the tongue cleaner; the sputum very adhesive. Crepitation was heard over the whole of the back of the right lung, with loud bronchial breathing at the upper part. A blister was applied, and he was ordered beef-tea.

March 31st.—The pulse was 76; the tongue clean; the skin moist, cool, and free from perspiration; the sputa were frothy and not discoloured; he had slept well; the crepitation at the back of the lung was less fine than before. He was ordered to omit the antimony, to take an effervescing mixture, with ipecacuanha wine, and to have a mutton-chop for dinner.

April 3rd.—He had slept well, and complained of no pain. The pulse was 90, small and weak. There was less dulness at the back of the lung; opposite the scapula there was returning crepitation, elsewhere the crepitation was coarser. He was ordered carbonate of ammonia, with tincture of squills and sulphuric ether. He progressed favourably up to the 5th, when he complained of pain in the left side, and there was a return of fever. The pulse was feeble and irregular. There was pain on pressure in the epigastrium. Examination of the chest showed a fresh accession of inflammation of the right lung, and a double friction-sound was distinctly heard in the precordial region towards the base of the heart, with pleural friction-sound on the left side. A blister and a saline mixture were ordered; and beef-tea was substituted for the chop.

April 6th.—The pulse was quick and irregular; there there was no pain. The pericardial friction-sound was about the same, the pleural more extensive. He was ordered two grains of calomel and one of opium three times a day.

April 7th.—The pulse was 110, and irregular; there was slightly increased precordial dulness. Friction-sound was heard when pressure was made over the centre of the precordial region, not otherwise. The heart-sounds were faint; no endocardial murmur was heard. He was ordered to take the pills every six hours, and to have a blister applied over the heart.

April 10th.—The pulse was 110, and regular. No pericardial friction-sound could be heard.

April 12th.—The pulse was 112, regular, but weak.

The heart-sounds were faint, but free from murmur. Friction-sound was heard over the lower part of the left pleura. The lung symptoms had somewhat improved since the last report. The gums were sore. There was no increased dulness of the precordial region upwards, but there was distinct bulging, with dulness, in the epigastrium. The pills were omitted, and carbonate of ammonia and wine were ordered.

April 14th.—He had not slept; there was much anxiety of countenance. Pulse 128, feeble and irregular. The tumour in the epigastrium was very distinct. There was no pericardial friction, nor endocardial murmur. There was less dulness at the back of the right lung. He was ordered an increased quantity of wine.

He died on April 15th.

AUTOPSY.—There was about a pint of straw-coloured fluid in the left pleural cavity. There was no effusion on the right side, but some old adhesions existed. The left lung was crepitant throughout, and healthy, except that it was somewhat emphysematous along the margins and at the apex. The pleural surface, laterally and behind, was covered with a thin layer of recent lymph. The right lung was consolidated, but not firmly, at the lower part of its posterior lobe. The upper part was much engorged. Anteriorly, the lung was crepitant. No tubercles existed. The lateral and posterior surfaces of the lung were covered with a thick layer of old lymph. The pericardium contained a very considerable quantity of purulent fluid. The heart was contracted and pushed upwards. The reflected pericardium was much thickened with layers of unorganized lymph; the adherent pericardium was covered with a thick layer of the same material, which assumed, in parts, the form of little pendulous growths from the surface of the heart. In some places, adhesions, thick, but easily broken down, existed between the two layers of the pericardium. The greatest deposit of lymph had taken place near the base of the heart, and in some spots it was more than half an inch in thickness. The fluid in the pericardium had pressed down the diaphragm, and produced the bulging in the epigastrium noticed before death. The muscular substance of the heart was healthy; the valves and endocardium were quite free from disease; atheromatous deposit existed in the aorta.

It is possible that whatever treatment might have been adopted in this case, the issue would have been a fatal one; but, with my present experience, I am disposed to think that, had the pneumonia been treated on a somewhat more stimulant plan, the pericarditis might not have supervened; and further, that the administration of mercury, in the latter affection, had a prejudicial effect, and that stimulants should have been given from its commencement.

Let me call your attention to the following case, which will serve to show the dangerous symptoms which sometimes follow the exhibition of mercury, and the value of free stimulation under apparently hopeless circumstances.

James W——, a bar-tender, 23 years of age, was admitted into the hospital, under my care, on the 24th February, 1863. He had been ill for three weeks before admission, and had been confined to his bed for one week. He had suffered from pain in the chest and dyspnœa. He had been attended by a surgeon, and was under the influence of mercury. On admission, he complained of pain in the chest, hands, and feet. He said he had been much purged. The pulse was 120, very feeble, but regular; the skin was hot, and there was profuse perspiration; the tongue was dry, brown, and furred. There was an anxious expression of countenance, and pain on pressure over the heart. A loud friction-sound was extensively heard in the cardiac region. He was ordered six ounces of port wine, a linseed-meal poultice to the chest, and the following mixture: bicarbonate of potash, three drachms; acetate of potash, two drachms; tincture of opium, eighty minims; water to eight ounces: one ounce to be taken every four hours.

On the 25th he had not slept, and had been purged several times. The pulse was 124. He had taken opium and brandy to check the diarrhœa. He was ordered to take ten minims of laudanum every hour; and to continue the alkaline mixture without the acetate of potash. He was purged six times during the afternoon, and in the evening was very prostrate. He was ordered an ounce of brandy every three hours, in addition to the wine, beef-tea, etc. The alkaline mixture was stopped, and an astringent one substituted.

On the 26th the diarrhœa continued; the friction-sound was heard less extensively over the heart, but, still, it was well marked; the pulse had risen to 132. He was ordered half an ounce of brandy, with arrowroot, every hour. He took nourishment freely. In the evening he had some sleep, and appeared somewhat better. A blister was applied over the heart.

On the 27th the bowels had acted nine times; he had taken twenty minims of laudanum with chalk-mixture after each motion; he had also had eight ounces of port wine, brandy and arrowroot every hour, and two pints of strong beef-tea. The pulse had fallen to 120. The blister had risen, and he said he felt easier about the chest. There was increased dulness in the cardiac region, but no friction-sound was audible. He was ordered to take a grain and a half of opium, at bedtime, and to continue the astringent mixture, with ten minims of laudanum, only, to each dose.

On the 28th the pulse was 118; the bowels had acted five times. He had taken eight ounces of brandy, and six of port wine in the twenty-four hours.

On March 1st he had passed a good night. The heart was felt beating in the second left intercostal space; the cardiac dulness was increased; friction-sound was heard on pressure, not otherwise. During the night he became somewhat delirious; and on the following day the diarrhœa was increased. A grain of opium was ordered every six hours, and an opiate enema after each motion, instead of the astringent mixture.

On the 3rd there had been some delirium during the night. The bowels were more quiet, and he appeared much better; the tongue was becoming moist, the skin was cooler, and the pulse had dropped to 102.

On the 4th the pulse was 100; and on the 5th it had fallen to 92. He had passed only two motions. There was less dulness over the heart, and friction-sound was heard on pressure. He was taking two grains of opium at night, ten ounces of port wine, and six of brandy, daily, beef-tea, etc. On the 7th the bowels were quiet; the pulse was 80, and the tongue cleaner. The heart-sounds were clear at the base; no friction-sound was audible.

On the 9th he began to take quinine, and the stimulants were diminished to eight ounces of wine and three of brandy, daily, with chop, etc.

He progressed rapidly from this date. On the 18th the brandy was stopped, and the quinine increased in dose. On the 21st there was no increased dulness over the heart; no friction-sound was audible; the heart's action appeared quite normal; its beat was in the usual place, and the sounds were free from murmur. He was discharged, well, on the 24th.

This case presents some features of peculiar interest. The patient had been confined to his bed for a week before his admission into the hospital, and we may assume, perhaps, that the pericardial inflammation had existed for that period. He had been put under the influence of mercury, but no beneficial effects had been produced on the disease. The remedy, instead of controlling the inflammation, had given rise to severe diarrhœa, which had resulted in a condition of extreme exhaustion; and although opium was given largely after admission, it was several days before the action of the bowels was checked. During that time the patient's state was very critical.

The quantity of stimulants given was very large, and they produced the most beneficial effects. Under their influence the pulse fell, the tongue became moist and clean, the delirium ceased, and the pericardial effusion became absorbed.

I shall give you one more instance of the value of free stimulation in pericarditis; and the case will serve to show that alcoholic stimulants may be safely administered, in large quantities, to the comparatively young.

Mary C., aged fifteen years, a somewhat short, spare girl, was admitted into the hospital on January 27th, 1862. She stated that she had been ill about a month, during which time she had been confined to her bed. The illness began with pain in the chest. For a week before admission she had suffered from a cough. No treatment had been adopted.

On admission, there was a good deal of distress, and anxiety of countenance; the pulse was 146; the respirations were 40 per minute. There were coarse moist *râles* over the whole of both lungs, but no dulness existed. There was a thrilling movement of the chest walls over the heart, with heaving impulse. There was increased dulness in the cardiac region, with a loud rasping friction-sound heard all over it. The patient complained of pain in the left side, cough, sleeplessness, and inability to take a deep breath.

The urine was free from albumen, and contained abundance of chlorides. She was ordered half an ounce of port wine every two hours, strong beef-tea, a linseed meal poultice to the heart, and seven minims of laudanum with seven grains of bicarbonate of potash, every four hours.

On the 28th, she had not slept during the night, but had dozed a little during the morning; she had vomited everything she had taken. The pulse was 132. The cardiac dulness extended to the top of the second piece of the sternum, and one inch and a half to the right, and four inches to the left, of the median line of that bone. The pericardial friction-sound was heard as before. There was a loud systolic murmur heard towards the left axilla, with slight friction-sound in the right pleura. She was ordered eight ounces of port wine daily; half a grain of opium every night; and ten minims of aromatic spirits of ammonia, with ten grains of bicarbonate of potash, three times a day.

On the 29th, the pulse was 140; she had slept but little, and complained of great thirst, and more pain in the left side. The pericardial friction-sound was heard when pressure was made with the stethoscope, not otherwise. There was less impulse. There had been a good deal of frothy mucous expectoration. Moist sounds existed all over both lungs. She was ordered to have an ointment applied to the chest, consisting of two drachms of mercurial ointment, with three grains of powdered opium.

On the 30th, she said she felt better, and had less pain. The cough was troublesome, but the expectoration was less. The pulse was 134. The pericardial friction-sound was only audible on pressure. The endocardial murmur was heard at the back of the chest. There was pleuritic friction-sound towards the left axilla. She was ordered meat for dinner.

On the 31st, the pulse was 142; respiration 48. The pericardial friction-sound could not be heard; the systolic murmur was audible at the base, and at the apex of the heart, but not in the direction of the aorta. The bronchial symptoms were still troublesome. She was ordered a blister, to be applied over the heart, and some carbonate of ammonia, squills, and opium, to be given every four hours.

On February 1st, the cough and pain were less. No pericardial friction-sound could be heard. The dulness was about the same.

On the 3rd, she was improving. The pulse was 134. The pericardial friction-sound was audible on pressure at the base of the heart.

On the 4th, there was less dulness in the cardiac region, and the impulse was stronger. The friction-sound was heard on pressure. She was ordered two grains of quinine with squills, and seven minims of laudanum, three times a day, with half a grain of opium at night.

On the 6th, there was further improvement. The pulse was 132. There was no pain. *No pericardial friction-sound could be heard.* The heart-sounds were clear under the right clavicle; but there was a loud systolic murmur audible over the heart and towards the left axilla. The opium at night was omitted.

On the 8th, the pulse was 126. There was but little expectoration. The other symptoms were about the same.

On the 13th, the cough was nearly gone. She was improving much in appearance, and gaining flesh. The pulse was 120. There was a dry harsh friction-sound heard at the back of the right pleura. The lungs were resonant behind.

On the 15th, the wine was diminished to six ounces daily, and the quinine to a grain and a half, with squills and sulphuric acid, three times a day.

On the 20th, the patient was convalescent. She felt quite well, and was able to get about the ward; but as she was suffering from valvular disease, and general debility, she was not allowed to leave the hospital.

The two last cases I have related were of an unusually severe character; the constitutional symptoms were very urgent, and the amount of effusion which took place was large. The disease had existed for some time before the patients came under my treatment, and it may, perhaps, be said that great debility had necessarily resulted, which demanded the free use of stimulants.

To show, however, that stimulants may be given, not only, with safety, but with advantage, even in the early stages of pericarditis, I will refer you to the following cases:—

Thomas D., a seaman, aged 37 years, was admitted into the hospital on December 1st, 1862. He was a stout, muscular man. He said he had suffered from rheumatic fever twelve years before his admission, but from no subsequent illness. Six days before he was brought to the hospital, he was seized with a sharp pain in the epigastrium, dyspnœa, and a sensation of constriction across the chest.

When seen on December 2nd, he complained of pain, increased on pressure, in the epigastric region, of a jumping sensation about the heart, and of pain in the limbs. There was no tenderness over the heart, nor any increased dulness; the cardiac sounds were free from murmur, but faint. The pulse was 94, and regular; there was an anxious expression of countenance. He was ordered some Dover's powder at bed-time, an ether mixture to be taken during the day, and for food, beef-tea.

On December 3rd, the pulse was 88, and dicrotic in the right arm. The patient had been delirious during the night, and the pain and anxious expression remained. Friction-sound was audible at the lower part of the right pleura in front. There was no friction-sound over the gressed satisfactorily to recovery. The wine was stopped on the 22nd, and the potash on the 27th. He subsequently took quinine; and was discharged, well, on January 27th, 1863.

J. W., 52 years of age, a labourer, was admitted into the hospital on the 3rd of May, 1864. Five weeks before admission, after exposure to bad weather, he had pain and swelling in the joints, and, three days before admission, swelling of the legs appeared, and extended upwards rapidly. He had rheumatic fever when he was about 26 years old. The chest, he said, was not affected at that time.

On admission, there was no perceptible swelling of the joints, and no pain was felt, but the legs were very œdematous. The tongue was moist and clean, the urine high-coloured, and free from albumen. There was no pain in the chest, and no friction-sound could be heard over the heart. He was ordered 40 grains of compound jalap-powder, and a mixture of sulphuric ether, and chloric ether, with beef-tea, and four ounces of whisky daily.

On the 5th, the pulse was 92, and there was pain in the left side. A loud friction-sound was heard over the heart. A mixture containing acetate of potash, with squills and juniper water, was ordered, and a grain of opium three times a day; the stimulant was continued.

On the 6th, the pulse was 84; the friction-sound was well-marked.

On the 7th, the œdema was nearly gone. The friction-sound was less distinct; there was pain on pressure in the cardiac region, with slightly-increased dulness. The pulse was 108, and regular. A blister was applied over the heart, and an ounce of port wine was ordered every three hours.

On the 8th, the pulse was 100; on the 10th it had fallen to 80, the patient's aspect was improved, and he said he felt much better. The friction-sound was louder again, and there was less dulness.

On the 12th, slight friction-sound was heard. Quinine was ordered.

On the 15th, he was much improved, and very slight friction-sound was alone audible.

On the 19th, no friction-sound could be heard.

On the 23rd, he was up, and said he felt quite well. He was discharged from the hospital on the 3rd of June.

In both these cases the pericarditis was developed whilst the patients were under my own observation, and, from an early period of the attack, stimulants were given. Let me call your attention to some of the features of the two cases. In the case of Thomas D., the pericardial friction-sound was heard on the third day after admission into the hospital. At that time the pulse was not very quick, but there had been delirium on the previous night, for which I ordered some opium. On the second day after we first heard the friction-sound, the pulse became quick (112) and irregular, there was an increase of the inflammation of the joints, and there was evidence of increased pericarditis. It was on this day, at the time that the symptoms were so severe, that I gave the stimulants, wine and ether, together with opium and potash. You can judge of the result of this treatment from the notes of the case. The pulse fell and became regular, the appetite improved, and convalescence was soon established.

On December 11th, viz., at the end of the eighth day from the friction-sound being first heard, it had disappeared, and there can, I think, be no doubt that adhesion of the two surfaces of the pericardium had taken place.

With regard to the second case, that of J. W., we had no evidence of pericarditis when the patient was admitted, but rheumatism had existed for some time, and there was some dropsical effusion about the legs. I, at once, prescribed stimulants for him, viz., sulphuric and chloric ether, and four ounces of whisky, daily. Two days after admission friction-sound was heard, and then, in addition to the alcoholic stimulants, I gave a diuretic mixture. The patient progressed satisfactorily, but the pulse having risen to 108 on the 7th of May, I ordered 8 oz. of port wine instead of the whisky. The daily record of the case will shew you that, under this treatment, the amount of effusion was never large, that we had early symptoms of its decrease, and that on the 19th of May we had evidence of its complete absorption, with, probably, the adhesion of the pericardium.

Thus, in these two cases, in which a moderately stimulating mode of treatment was pursued from the early periods of the disease, none of those severe symptoms were developed, such as characterized some of the other cases which I have referred to, and we had an early convalescence.

There is one symptom, which is mentioned in the notes of the first of these two cases, about which I wish to say a few words, viz., the dicrotic pulse. I have often pointed out to you cases in which the pulse has had a double beat, a second feeble one, after the first stronger one.

The sphygmograph has shewn us that there is a dicrotism of the normal pulse, but this dicrotism is not perceptible to the touch, and it is only under certain conditions that it becomes so. Many opinions have been expressed with reference to its cause, and I think it is important that you should understand, not only its mode of production, but also its import, as a clinical fact, when it becomes strongly marked.

An opinion has been expressed by Dr. Marey, that the dicrotism of the pulse in the radial artery is due to the "echo" of a wave of blood, which is reflected at the bifurcation of the aorta, by the spur of the two iliac arteries.

This view, Dr. Marey has endeavoured to prove, by an appeal to experiments made in an apparatus, resembling somewhat the aorta, having branches to correspond with the iliac vessels, and with those of the upper extremities.

I have no doubt that the phenomenon is the result of a reflux, or "echo," as Dr. Marey has expressed it, of the sanguineous wave; but I cannot agree with the view that this reflux is produced at the bifurcation of the aorta.

The condition under which the phenomenon is most marked is *a feeble tension of the arterial system.* The more feeble the tension, the more marked is the dicrotism of the pulse. The strongly-marked dicrotic pulse is, in fact—and this is the most important practical point in connection with the subject—essentially a pulse of debility. My own view is that it is caused by *a reflux of the blood produced by the closure of the aortic semi-lunar valves,* and not by the "echo" reflected at the bifurcation of the aorta. When there is feeble tension of the arterial system, when the walls of the arteries are yielding, and these vessels are not fully distended by their contents, the sudden closure of the aortic valves gives an impulse to the blood,—a shock, in fact, which is more or less felt, especially in the vessels arising from the arch of the aorta. The reason why the shock is not felt in the lower extremities is, probably, in consequence of the curve which the aorta makes, and which, it appears to me, would prevent the transmission of the shock, except in some extreme cases, where the arterial tension is very feeble. I expressed this view of the cause of the dicrotism of the pulse several years ago, before the sphygmograph had taught us what we now know of the nature of the pulse.

But as to the import of the strongly-marked dicrotic pulse, such as can be appreciated by the finger. Whatever be its exact cause, it is, clinically, very interesting and important. As I have already remarked, it only occurs where there is feeble arterial tension, and it is always characteristic of debility. It is not characteristic of any particular disease, but it is indicative of a pathological condition, which comes on in a great variety of diseases; and although, when it exists, there may be other symptoms to guide us to a right judgment of the case, the presence of the dicrotic pulse forms an additional element in the diagnosis, and affords an additional indication as to the line of treatment to be adopted.

It is not very often that an opportunity is afforded us of following a case of pericarditis, where recovery has taken place, to the death of the patient some time after, so as to be able to ascertain the exact morbid condition which has resulted from the disease. Such an opportunity, however, occurred in the following instance.

Thomas S., aged 17 years, a labourer, was admitted into the Hospital, under my care, on the 23rd April, 1861. He had been the subject, he said, of five separate attacks of rheumatic fever during the previous eighteen months, and on one occasion his chest was much affected. About five days before admission, he was seized with pain over the hips and arms, and, two days afterwards, with pain at the heart. The latter pain had gone on increasing.

On admission, he complained of pain in the knees and hands, as well as in the epigastrium and over the heart, when a pressure was applied. There was pain also in the left side, on taking a deep inspiration. The respiration was thoracic, the skin moderately cool, and there was expectoration of frothy mucus. The lungs were resonant; there was more than the usual extent of dulness in the cardiac region, with increased impulse of the heart. The impulse was felt below, and to the left of the left nipple. There was a loud systolic flesh. He was kept in the hospital till July 3rd. When discharged, he was much improved in health, but he suffered from the symptoms of hypertrophied heart, with valvular disease. The systolic murmur was very loud, and audible all over the front of the chest, and on the left side, behind; there was increased impulse of the heart, and increased cardiac dulness.

From the course which the inflammation of the pericardium took in this case, and from the manner in which the friction-sound disappeared, I concluded that adhesion of the two portions of the pericardium had occurred; and this opinion was afterwards found to be true, as will be seen by what follows.

Some months after his discharge, the patient was readmitted into the hospital, under Dr. Collingwood, suffering from general dropsy, and he died in December. The following were the *post-mortem* appearances presented by the heart:— The pericardium was universally adherent. The heart itself was large, and weighed two pounds avoirdupois. The walls of the left ventricle were much thickened, and dilated. The aortic valves were rough, and thickened with deposit. The mitral valve was also thickened, and so contracted as barely to admit two fingers through the orifice.

Having alluded to these cases, I shall now briefly refer to the conclusions at which I have arrived, from clinical observation, with regard to the general principles of treatment in acute pericarditis.

My belief is, that it is rarely, if ever, desirable to give mercury, except, occasionally, as a purgative; that it has no special power of controlling the inflammation, and that, in giving it to the extent of producing salivation, you would lower your patient's strength, and increase the danger of a fatal issue. There may be exceptional cases in which it should be used, but I am now speaking of such cases as are generally met with, and of my own experience in the treatment of the affection.

With regard to opium. Great benefit is, in my opinion, derived from the regular administration of opium, in grain doses, repeated every three, four, six, or eight hours. The solid form of the drug is, I think, generally, the best to give. You need not be deterred, as some are, from giving opium in acute rheumatism, under the impression that it has a tendency to check the secretions, and thus to retain the rheumatic poison in the system. It seems to me to have, as a rule, no such influence; and it is very remarkable how regularly the bowels of rheumatic patients will act, although they are taking several grains of opium a day. This is a point on which I have often had to speak to you in going my rounds.

Next, with regard to stimulants. I think that, in most cases of pericarditis, it is advisable to give a moderate quantity of stimulants from an early period of the disease. The quantity must be measured by the character of the pulse, and the general condition of each patient. If there be any tendency to delirium, or if the pulse, previously regular, become irregular, intermittent, or dicrotic, the quantity of stimulants must be increased, and it will generally be found that, under their influence, these symptoms will disappear. It is of great importance, in all these cases, to watch the patients very carefully, so as to anticipate and prevent an outbreak of delirium, by meeting the early indications of its approach by suitable measures.

Whatever may be the nature of the rheumatic poison, there cannot be a doubt that, in rheumatic cardiac inflammation, alcoholic stimulants are often very beneficial. Wine answers very well in some cases, but where there is much depression, brandy should be given. Again, in cases of rheumatic fever affecting only the joints, but marked by a very rapid pulse, and indications of debility, I have given wine freely, with great benefit to the patients.

I have already drawn your attention to the case of John M., in speaking to you of rheumatic pneumonia. This man benefited very much from the free use of stimulants in an acute attack of articular rheumatism, and you frequently see me give smaller quantities of alcohol in rheumatic fever, if I find the pulse very quick and feeble.

But although I can confidently recommend you to administer stimulants in such cases as I have referred to, I, by no means, wish you to infer that they are proper remedies in acute rheumatism generally. You know that they do not form an element in my ordinary treatment. When acute rheumatism attacks the heart, I consider it of the utmost importance to watch for any signs of flagging action of the organ, and to exhibit stimulants in proportion to their urgency. In most cases of rheumatic pericarditis, it is probable that the substance of the heart is, more or less, affected, and that stimulants are beneficial to the organ in its weakened state, just as they are so when it is enfeebled from chronic disease.

With regard to other remedies in pericarditis. I usually prescribe bicarbonate of potash, and I have some confidence in its remedial power. I have now used it in a large number of cases of acute rheumatism, and from the generally favourable termination of these cases I see reasons for continuing the practice. Certainly, whether from the use of the remedy, or from some other element in the treatment, or whether from some cause of which I am ignorant, I have seen, of late, fewer cases of heart complication in rheumatic fever than formerly.

I have never taken blood by venesection in pericarditis, and I have rarely found it necessary either to cup or leech;

and although there can be no doubt that great relief to pain often follows the local abstraction of blood, I have generally found that the same end may be obtained by the administration of opium.

Counter-irritation by blistering I usually resort to, but not at the commencement of the disease. I believe it has a reflex influence on the inflamed blood-vessels, that it modifies their action, and promotes absorption of effused matters.

The application of warm linseed-meal poultices to the chest I consider an important adjunct in the treatment of this, as of some other acute thoracic affections. These poultices usually afford great relief to the patient, and should be used from the very commencement of the disease. It is desirable to expose the surface of the chest as little as possible; frequent examinations are objectionable, and care should be taken on renewing the poultices that a chill is not produced.

The practice of wrapping up the patient in flannel is, I think, a good one.

I always allow nourishment, beef-tea, milk, etc., throughout the disease, and solid food, as soon as the state of the appetite enables the patient to take it.

CHAPTER III.

FATTY DISEASE OF THE HEART.

(CLINICAL LECTURE.)

GENTLEMEN,—I wish to call your attention to-day to the subject of fatty disease of the heart.

Fatty disease of the heart occurs in two forms: first, as fatty deposit upon the heart, and between its muscular fibres; and, secondly, as fatty degeneration of the muscular fibres themselves.

Setting aside, for the present, the first form of the disease, let me direct your attention to the second form. It is only of late years that the exact anatomical characters of this affection have been known. It is true that Laennec, and previous writers, had recognized the malady, and that Laennec accurately described the naked-eye appearances presented by the fatty heart. But it was not until microscopical examination was made, that all doubt was cleared up as to the actual changes which the muscular fibre undergoes.

Amongst the most important researches into the nature of this affection must be mentioned those of Dr. Quain, which you will find in the 33rd volume of the 'Medico-Chirurgical Transactions.'

I must refer you to that paper, and to your systematic works, for a description of the appearances which the fatty heart presents; I have, on several occasions, shown you specimens of it under the microscope. It must be sufficient for me to say that, in the disease, the characteristic transverse striated appearance of the muscular fibre disappears, and there is deposited, in the substance of the fibre itself, a number of small oil, or fat globules. Thus you see that the contractile element of the heart is, more or less completely, destroyed, and, in its place, is deposited a substance which possesses no contractile power.

To such an extent does this change sometimes occur, that we are astonished, on examining the heart after death, that it has been able to carry on its function at all. This destruction of a large portion of the muscular fibre of the heart is, I think, sufficient to prove that the force, required to circulate the blood throughout the body, is much less than many physiologists have supposed.

But, although recent observations have made us familiar with the morbid anatomy of this disease, it must be confessed that great obscurity hangs over its pathology. Of the intimate nature of fatty degeneration of the heart, and of the causes which lead to it, very much remains to be discovered. Still, I think, we have important evidence on both these points, evidence which affords us valuable indications as to the treatment which should be adopted in the disease.

There are two conditions under which fatty degeneration shows itself, which have important bearings on, and perhaps a close relation to, each other. I mean fatty degeneration of the heart, and atheromatous deposit in the arteries.

It is true that fatty disease of the heart is met with when no atheromatous deposits are found in the blood-vessels; but it is also true that, in a very large proportion of cases, the two conditions are found existing together.

There is one very important point in connection with this disease, to which I wish specially to draw your attention, viz., its frequency in old age. In fact, it seems to be a very constant accompaniment of advanced years.

One of the principal changes, occurring in the body at the approach of old age, is the conversion of certain tissues into fat. In many people, as age advances, there is a tendency to the deposition of fat on the surface of the body, and around, and upon certain internal organs. This constitutes ordinary obesity. But there is, further, a tendency in certain organs, especially in the heart and blood-vessels, to undergo fatty degeneration—for fatty matter to become deposited in the place of normal tissue. These changes are not, necessarily, connected with old age, but it is a rare thing to examine the body of a person, who has reached advanced years, without finding them present, to a greater or less extent.

Now, this alteration of tissue is, undoubtedly, a degeneration, not only because fat is mechanically useless, and of lower organization than the tissues which it replaces, but because the conditions, under which it is deposited, partake of the nature of death. It is a slow annihilation of the structure, and, consequently, of the function of the organ which it attacks.

Although I am not disposed to take a chemical, or a mechanical view of the nature of these fatty changes, I must remind you of some conditions under which alterations of tissue, partaking much of the nature of these degenerations, may be produced.

It has been shown that fatty degeneration of the placenta takes place towards the end of utero-gestation, at a time when the organ is becoming relatively old. Further, there is the well-known fact of the conversion of dead flesh into adipocere, and the experimental demonstration of Dr. Quain, that a piece of healthy muscle, placed in a jar and kept moist, without access of air, will, in a few weeks, present the appearances of fatty degeneration.

Now, I would not attach too much importance to the changes to which I have last alluded; for, after all, changes occurring in animal tissues after death, although they may, to some extent, serve to explain certain changes occurring during life, yet, they may be altogether of a different nature; and, moreover, the most recent experiments on the subject, those of Dr. Ormerod, tend to show that, although, when a piece of muscle is placed in a jar and kept moist, it presents, after a short time, an appearance very like that which fatty degeneration presents in its early stages, yet, the subsequent changes do not at all correspond with the subsequent changes of the diseased muscle; and, from a careful examination of this subject, I think we must conclude that the change which takes place in a dead muscle, its conversion into adipocere, bears no relation to fatty degeneration—the one is chemical, the other vital.

But besides the changes which take place in dead flesh, which I think we may pass over as having no bearing on our subject, and independently of alterations which take place in the placenta, and also of those, far more important ones, which may be regarded as characteristics of old age, fatty degeneration of muscular fibre may be induced by other conditions, and these have, I think, a very important bearing on our subject.

It is well known that, whenever a voluntary muscle—a muscle of one of the limbs—is placed in a condition of rest for a long time, it undergoes not simply atrophy, but fatty degeneration. Let me refer you to a case which fell under my own observation.

I made a careful examination, some years ago, of the muscles connected with the knee-joint of a limb which had been amputated. The limb, in consequence of disease of the knee-joint, had been fixed in one position—that o extreme flexion—for a considerable time, during which the action of the muscles of the calf had been, more or less, in abeyance. The gastrocnemius had all the appearance, to the naked eye, of fat. The soleus was slightly red, but evidently fatty. On placing portions of these muscles under the microscope, I found that the gastrocnemius was almost entirely made up of fat-globules; that, in fact, it was in a very advanced stage of fatty degeneration. The soleus was also fatty, but to a less extent than the other muscle.

In the microscopical appearances presented by these muscles, I could find nothing which would enable me to distinguish them from those of fatty degeneration of the heart; and I think the changes which I observed, and which are the ordinary ones met with in paralyzed limbs, are very closely indeed allied to, if they are not even identical with, those which take place in the fatty heart.

There was a difference, as I have observed, in the two muscles I examined — a difference not without its interest. The gastrocnemius was the more degenerated. This muscle, from its connections, and from the position of the limb, was probably altogether deprived of action, whilst the soleus was brought into play, to a certain extent, in extension of the foot. This circumstance will serve to account for the anatomical differences observed in the two muscles.

Now, you must bear in mind, that, whilst these muscles were undergoing this fatty degeneration, they were still part of the living limb, they were regularly supplied with blood, and there was no impairment (except a local one) of their nervous energy—they were simply at rest—their physiological action was suspended, and this was sufficient so to impair and prevent their nutrition, that their highly organized contractile element became converted into a substance of much lower organization, and one which was utterly useless for the function of the muscle.

It cannot be said that the change, taking place in these muscles, was simply a chemical change; it was essentially a local impairment of nutrition; and there is little doubt that, could those muscles have been gradually brought into action again, the integrity of their structure might have been restored.

But there are some other circumstances to which I wish to draw your attention, as tending, I think, to throw some light on the nature of the disease. It is well known that in animals fattened for the purpose of exhibition—animals which are allowed no exercise, and an unlimited supply of food—fatty degeneration of the heart is very frequently met with. Further, I have frequently examined the hearts of persons who have been very fat, and I have almost invariably found, not only a large deposit of fat upon the heart, but that its muscular fibres had undergone fatty degeneration. Now, possibly, this change may in some measure be due to the general predisposition to obesity, but I am disposed to attribute it, mainly, to another cause.

Obesity has a great tendency to lead to inactive habits, a circumstance which tends further to increase obesity. We are all familiar with the flabby condition of the muscles of the fat man, a condition which shows that they have been imperfectly used, and I have given you illustrations of the effects which follow extreme inactivity, and absolute rest of muscles. Now, you must bear in mind that the heart has a structure similar to that of ordinary voluntary muscles, and just as we see that fatty degeneration of the latter may take place when they no longer perform their functions, so, I think, we may fairly expect that a like process will follow in regard to the heart, under circumstances of a similar nature. For although, from an early period of embryonic existence to the moment of dissolution, the function of the heart is never arrested, yet it may be imperfectly performed. Inactivity of the general muscular system means inactivity of the heart itself; and in those who lead a sedentary life the heart is underworked, and may, therefore, be under-nourished, and become degenerated.

Further, there cannot be a doubt, I think, that where there is obesity, with a deposit of fat upon the heart and between its fibres, there exists an additional source of inactivity—comparative rest—of the organ. The weight and pressure of the fat upon its fibres must seriously impair the vigour of its contraction. And although we do occasionally meet with cases in which, whilst there is a large deposit of fat upon the heart, the fibre itself is unchanged, still, these are exceptional cases, and are mostly found in persons who, in spite of their fat, have led an active life, and have been much in the open air. In the case of obstruction to the circulation from valvular disease, the muscular substance of the heart becomes hypertrophied; but in the case of fatty deposit it is the muscular substance itself which is over loaded, and over weighted, and no such compensating action, as that which occurs in valvular disease, can be expected. The action of the heart is repressed in the one case, whilst there exists a stimulus to its increased action in the other.

I can call to mind several cases which would serve to illustrate these views—cases of persons who have led an inactive, a sedentary, or an indolent life, who have lived but little in the open air, and who have become the subjects of fatty degeneration of the heart.

Let me say a few words in regard to the physical signs, and symptoms of the disease. There is nothing peculiar about the physical signs, nothing more than you will meet with where the heart is weakened from some other cause. The impulse is feeble, and, in advanced cases, it becomes imperceptible, both to the eye and hand. The area of cardiac dulness does not undergo any change, unless there is some alteration of bulk from some cause independent of the fatty disease.

The sounds of the heart are faint, especially the first, which, in some cases, becomes almost extinguished. This rarely happens to the second sound.

These are just the physical signs which you might expect, when the heart, having undergone no change of bulk, has had a portion of its muscular fibres converted into fat. The organ necessarily contracts with but little force, and hence the feeble impulse; hence, also, the faint first sound, from the fact that the auriculo-ventricular valves are not as forcibly stretched by the impelled blood as in health.

Although there is nothing in the physical signs that will help you much in forming a diagnosis in the disease, there are symptoms which afford most important aid in this respect, and which, taken together with the physical signs, enable us often to speak with much certainty.

There is great variety in the pulse in the affection. It is weak, soft, compressible; generally, but not always small; sometimes intermittent, irregular, and unequal. It is, occasionally, very slow, even as low as 20 to 30 in a minute, but, in the majority of cases which I have met with, it has had a medium frequency, and sometimes the pulse is, not only not small, but, on the contrary, of very good volume. I made, several years ago, a *post-mortem* examination of a gentleman who had died after a few days' illness, and who had long been supposed to have a fatty heart. Throughout the three days, in which he lay in a semi-comatose condition, his pulse was of tolerably good force, and of good volume. I found, on examination, that the heart had undergone fatty degeneration to a great extent, and that there was also a large deposit of fat upon it, as well as a generally obese state of all other parts of the body. I attended, not long ago, a lady, who died of dropsy, who, for some years, had been getting very fat, had taken little exercise, and had suffered from many of the symptoms of fatty heart. She had no valvular disease. The heart-sounds were free from murmur, but faint. The pulse was of good volume, very soft and compressible, and quite regular. I could not obtain a *post-mortem* examination, but I have no doubt that extensive fatty disease of the heart existed.

I allude to these cases in order that you may be aware that, because your patient has a moderately full pulse, you must not conclude that he has not a fatty heart. I believe, in these instances, there is a relaxed condition of the arterial walls, a condition which allows of distension even from a feeble contraction of the heart, for, whilst the pulse is of good volume, the impulse may be very feeble. Further, the persons, in whom I have observed this condition, have been of full habit, and the blood-vessels were consequently well-filled.

The pulse is very much influenced by the state of the digestive organs, a remark which will apply to other diseases of the heart. Some patients have an irregular pulse after every meal, the pulse becoming regular as the stomach gets rid of its contents. Again, a flatulent condition of the stomach will often bring on irregular action of the heart, and the occurrence of this symptom may be the first thing that attracts the attention of the patient to his malady.

Certain disturbances of the function of respiration are very commonly met with in this disease. There is dyspnœa on exertion, and especially on going up hill, or upstairs, and you will observe that patients often stop to take breath. There is a peculiarity which I have frequently observed in well-marked, uncomplicated fatty disease, which other authors have drawn attention to, viz., frequent sighing. Some years ago, a medical practitioner was brought to me by his brother for examination. He was supposed to have heart-affection. He had many of the usual symptoms of the disease I am describing, and, amongst other things, I learned that he was constantly sighing. Indeed, whilst he was in my room, he sighed frequently. I did not hesitate to express my opinion that he was suffering from fatty heart, and to give an unfavourable prognosis. A few weeks after I saw him, he died.

Sometimes the dyspnœa assumes a paroxysmal character. Patients will wake up in the night in great alarm, and gasping for breath. The symptoms you might, possibly, mistake for those of asthma. But you must be careful not to commit such an error. If you give your patient, suffering from cardiac apnœa—for that is what the attack is—remedies of a depressing character, you may paralyze the feeble heart. Stimulants are required; whereas, in cases of asthma, the exhibition of chloroform, or the smoking of stramonium, may give immediate relief. The explanation of these attacks, of the dyspnœa, and of the sighing, is in the feeble heart, which is unable to send the blood to the lungs sufficiently fast. The breathlessness is from want of blood, not from want of air.

The occurrence of syncopal attacks is by no means infrequent in this disease. The attack may be slight, and pass off without loss of consciousness, or insensibility may result. These attacks are often brought on by some error of diet, or by something which has taxed the powers of the heart too much—as anxiety, mental, or physical labour. The symptom is a very serious one, for it indicates a very feeble condition of the heart. It is one of the chief causes of sudden death in the disease, a mode of death to which patients with fatty heart are very liable—a circumstance which I shall have to allude to again.

Some patients suffer from pain of a very severe character, whilst in others, the symptom never occurs. The pain sometimes extends very widely over the chest, coming on in paroxysms, constituting, what is called, angina. It very frequently shoots down the left arm and to the left scapula, and sometimes down the right arm. The occurrence of pain in the left side, extending down the arm and reaching, as it often does, to the fingers, should always cause you to examine the heart very carefully, for this is, sometimes, amongst the earliest symptoms of the disease. I have also met with another symptom in connection with the left arm, in some cases of fatty disease of the heart—an occasional numbness, and a blanched appearance of the extremities of the fingers. Both these symptoms, the pain down the arm, and the altered circulation, are reflex phenomena of a very important and interesting character. Pain is sometimes associated with a syncopal attack, and the two phenomena constitute the syncope anginosa. You may recollect the case of John W., who was in the Hospital in October, 1866, and who suffered from attacks of this kind. Let me refer you to the notes of his case.

The patient was sixty-nine years of age, a joiner by trade, and he was admitted into the Hospital on the 26th October, 1866. He said he had been ill for two years, and that he suffered from attacks of "cramp in the stomach," and shortness of breath. The pain, he said, was very severe, and extended over the left side, and down the arm. The attacks usually came on during the day. In other respects he considered himself pretty well.

On examination of the chest we found the percussion and breath-sounds normal. The sounds of the heart were faint, but free from murmur; the impulse was scarcely perceptible. The pulse was feeble, regular, 76 per minute. The patient had a sallow aspect, and was not fat. He was ordered some brandy, and a mixture containing sulphuric ether and ammonia, and, on the 23rd, he was put on ten minims of tincture of iron three times a day. On the 30th he had one of his attacks, at 10 a.m. He was seen by the house-surgeon. The attack lasted about ten minutes, and is thus described: "Irregular action of the heart; pulse feeble, but perceptible at wrist; pain in the pericardial region and epigastrium; pain shooting down both arms. The attack came on whilst he was walking in the lobby, and he managed to reach his bed. He was somewhat blue at first, and then he broke out in a cold, clammy perspiration. He was never insensible." He had another fit the same day at 11 a.m., which came on whilst he was in bed, and lasted about ten minutes. He continued free from an attack till the 8th November, when he had one about 1.30 p.m., which lasted about the same time as the former ones. He had fits on the 12th, 17th, 19th, and 20th, and on the 27th he was discharged.

Now, the symptoms which I have just described constitute an attack of what is called "angina pectoris," and, in this instance, I have no hesitation in saying that I believe they were connected with a fatty condition of the heart.

There is another symptom, to which I must draw your attention, which is met with in this disease, viz., coma. It is rare to meet with this symptom, except in cases where the disease is advanced. Patients are suddenly seized with symptoms of an apoplectic character, except that there is very rarely stertor, and paralysis does not follow. They fall, become quite unconscious, the face is pale, and the surface of the body cold; the pulse is very feeble, and there are usually no convulsions, although there may be some twitchings, and convulsive movements. On the return of consciousness, the patients remain in a lethargic condition for some time, from which they gradually rally, but sometimes the coma kills them; in 83 cases tabulated by Dr. Quain, in the paper I have referred to, seven died of coma.

Independently of these symptoms, there is, about the patients who suffer from advanced fatty disease of the heart, a peculiar aspect, which can scarcely, I think, be mistaken. There is a pallor, a sallowness of the skin, and a flabbiness, which, I think, no other condition presents. Further, they are unequal to much exertion, and soon become fatigued, and out of breath; they are apt to suffer much from flatulence, of a very distressing kind, and very difficult to cure. They very frequently take a desponding view of their own case, and, especially if they have had any syncopal, or semi-apoplectic attacks, imbibe a notion that they will some day die suddenly.

But whilst I tell you of all these symptoms, which may be, and are, constantly met with in the disease, I must also remind you that many of them may be absent, even when the disease is far advanced; or, at least, they may not exist to such an extent as to attract the attention, even of the patient himself.

We occasionally hear of persons dying suddenly of this disease, without, as it is said, any previous symptoms having occurred; whilst, on *post-mortem* examination, the heart is found extensively fatty. The following is a case in point:

A gentleman, between 60 and 70 years of age, met with a slight accident, whilst driving in his carriage. He was confined to the sofa, and attended by his surgeon. His previous health had been, as he thought, good; in fact, he boasted that he had not been unwell for many years, and had never had occasion to consult a physician. Five or six days after his accident, he was turning on his sofa, and died suddenly. An examination was made, and his heart was found extensively fatty.

Now, cases of this kind are occasionally met with, but

I must express a doubt whether fatty disease of the heart ever reaches such a stage as to cause sudden death, without producing some symptoms which ought to lead to a recognition of the malady, or at all events, to a suspicion of its existence. And, in the case I have referred to, probably, a careful cross-examination of the patient would have led to the detection of some, perhaps, unequivocal symptoms. It very often happens, in other organic affections, that patients go on for a long time neglecting and disregarding minor symptoms, and even imagining themselves in perfect health, until some accident, or the occurrence of some acute attack, summons us to their bedside, and we find evidence of long-standing organic disease, which has been gradually undermining the health; and then we learn, on questioning our patient, that certain symptoms, which would, at once, have pointed out the nature of the disease to us, have occurred.

But still, the occurrence of cases such as I have alluded to, shows how insidious this fatty disease of the heart sometimes is, and how careful we should be to study all those external symptoms of the malady, which may be so easily recognized. And this leads me to say a few words in reference to the existence of the "arcus senilis," or fatty degeneration of the cornea, as a symptom of fatty degeneration of the heart.

I have, of late years, directed my attention to this condition of the cornea, and I have come to the conclusion that, as a symptom of fatty degeneration of the heart, it is by no means of certain import. Its presence does not necessarily prove that the heart is fattily degenerated, for it may be, simply, a local degeneration; nor, on the other hand, does its absence prove that no degeneration of the heart exists. Still, as an indication of a tendency to fatty degeneration, it is an important symptom, and, undoubtedly, it is frequently found in connection with diseased heart. And I would observe that the absence of the "arcus" must not be hastily assumed; for, many persons who, at first sight, may appear to have no signs of it, are yet found, when the cornea is fairly exposed, to have the upper portion more or less marked.

Fatty degeneration of the heart, to a greater or less extent, is, I believe, a far more frequent affection than is generally supposed; indeed, it is rare, in affections which impair the nutrition generally, to find the heart, after death, free from fatty change, if it be carefully examined under the microscope. In hearts which have been the seat of inflammation, either of the pericardium or endocardium, fatty degeneration of the muscular fibres is generally found. I have never examined a heart in which there was an adherent pericardium, the result of previous acute inflammation, without finding the muscular tissue extensively fatty. Again, in cases of long-standing valvular disease, whether of acute or chronic origin, I have rarely failed to discover, under the microscope, evidence of fatty change.

There can be no doubt that acute, or chronic inflammation, attacking the membranes or the substance of the heart, favours the subsequent degeneration of the muscular fibres. I shall have to point out to you, when I come to speak of valvular disease of the heart, that the muscular degeneration constitutes, in my opinion, the most serious part of the affection; that, as long as the muscular fibres retain their vigour, their power of contracting with force, there is but little danger to your patient; but that, as soon as their vigour is diminished by degeneration of their structure, the secondary symptoms, dropsy, etc., begin to show themselves, and the liability to sudden death, which always exists in heart disease, is increased.

Fatty disease of the heart is very frequently met with in old standing cases of gout. The pale, flabby look, which characterizes many gouty people, is an indication of the existence of fatty disease. A knowledge of the tendency to heart degeneration, in gout, should make us careful in the management of this affection, and it shows us the danger of suddenly withdrawing from gouty patients their habitual stimulants.

Again, it cannot be doubted that fatty disease of the heart constitutes a very serious complication in numerous other affections. If an acute disease, such as pneumonia, fever, bronchitis, erysipelas, or delirium tremens, attack a person with a fatty heart, it adds much to the danger of the disease, and to the difficulty of treating it. It is not always possible to ascertain the existence of this condition of the heart in such cases, but the fact should teach us to use great caution in the administration of depressing remedies, when, from the age of the patient, or the previous habits of life, there may be a probability of the nutrition having been imperfect. I think that, in many cases of death from delirium tremens, a fatty condition of the heart has been the real cause of the fatal issue. And, again, in cases of fever, it seriously increases the danger of the attack. I have seen cases of this disease, in which I have had no doubt of the existence of a fatty heart, and in which, in spite of every effort to rouse the circulation, the patients have sunk exhausted from mere syncope.

Not only does this fatty disease of the heart complicate the various acute affections with which the physician has to deal, and render recovery from them more difficult, but, further, it increases the liability to a fatal issue in all serious accidents, and, especially, in all capital operations. It is well to bear these circumstances in mind, because blame may sometimes be supposed to lie at the door of the practitioner for the unsuccessful issue of his treatment, whereas the result may be due to the existence of a heart unable to perform its function.

I believe that fatty degeneration of the heart is not very constantly met with in phthisis. I have not made many careful microscopical examinations of the heart in this disease, but the general appearance of the organ, as seen by the naked eye, would lead me to infer that fatty degeneration is only occasionally found. There is one chronic affection, however, viz., Bright's disease of the kidneys, in which the heart very frequently undergoes fatty change.

There is another pathological condition of the body to which I wish to call your attention, in connexion with this subject. It has been observed that, in cases of "arcus senilis," and fatty degeneration of the heart, ossification of the cartilages of the larynx, of the rings of the trachea, and of the cartilages of the ribs is often met with. I have frequently noticed that the cartilages of the ribs have become hardened, and in part ossified, at a somewhat early period of life; and, contrary to the expressed opinion of some pathologists, I pointed out, several years ago, that the rings of the trachea often have ossific deposits in them, long before the approach of old age. I believe that this degeneration of the costal and tracheal cartilages is of a nature closely allied to fatty degeneration. Let me refer you to a case bearing on this point. Some years ago a man was admitted into this hospital, under my care, suffering from severe hæmoptysis. He died a short time after admission. At the autopsy we found a large quantity of blood in the left pleura, and in the lungs. There was an aneurism of the thoracic aorta, which had burst into the left lung and pleura. The aorta was atheromatous. The muscular fibres of the heart, although to the naked eye they appeared healthy, had undergone fatty degeneration to a considerable extent; and there was osseous deposit along the centre of most of the costal cartilages. Here, then, there was a combination of fatty disease of the heart, atheroma of the arteries, and ossific degeneration of the cartilages of the ribs.

But little is known as to whether a predisposition to fatty disease of the heart is transmitted from parent to offspring — whether, in fact, the disease is hereditary. My own experience affords no evidence on this point.

The disease is essentially one of middle, and advanced life. It is rarely met with in young persons, unless associated with some pericardial, or endocardial disease. I have seen it in an advanced state in a man, twenty-five years of age, who had valvular disease of the heart. It seems to be more frequent amongst men, than amongst women, and although it occurs in all classes of society, it is considered by most physicians to be more prevalent in the upper, and middle classes, than in the lower. Certainly my own experience accords with this, as far, at least, as regards the pure, uncomplicated form of the disease. Nevertheless, a careful examination of the hearts of patients who have died in hospital, reveals a large proportion in which there has been a fatty change.

And now let me say a few words as to the treatment of this affection. I think it is only in the early stages of the disease that we can hope, by remedial measures, to effect a cure —I mean, that we can hope, so far to improve the nutrition of the heart, that its structure may be restored to a healthy state. When the disease is more advanced, we may be able to check its progress, and this must be considered a satisfactory result; but, where extensive degeneration of the muscle has taken place, all we can do, is, to mitigate the symptoms which arise, and endeavour to keep up the flagging action of the heart.

Now, a consideration of this should lead you to study the early symptoms of the disease, so that you may recognize it at a time when remedial measures are most likely to be of service.

But what are the measures which you should adopt, when you feel tolerably sure that you have to deal with a case of this disease?

You must resort to all those measures which have an influence in improving the nutrition of the body. If your patients are corpulent, and of sedentary habits, — and I have told you how frequently such persons are subject to the disease, — you must induce them to lead a more active life, and to be a great deal in the open air; you must put them on a diet which is nutritious and not bulky, with a fair amount of stimulants. I have great confidence in open-air exercise in these cases, but the exercise must be judiciously taken. Fatigue should always be avoided, and all violent exertion should be rigorously forbidden. The exercise may be increased from day to day, and as the general muscular system improves, so will the heart, often, improve. You should select for your patient an elevated spot as a place of residence, with a dry bracing atmosphere, and if he is able to drive about on the hills, so much the better. A climate like that of Harrogate often proves very beneficial in these cases. All excitement must be avoided. The depressing influence of care and anxiety has a very prejudicial effect.

You may prescribe any tonic medicines which will agree with your patients, in this affection; but there is only one substance in which I have any real confidence, as possessing a curative action in the disease, viz., iron. I believe that, in iron, we have a substance, which, if taken in small doses for a long time, is capable of restoring to an enfeebled and fatty heart, a good deal of its vigour, and possibly of its structure. But, in order that it may do good, it must be continued not only for weeks, but for many months, and even for years, being omitted, from time to time, as the state of the digestive organs may require. There is probably no better preparation than the tincture of the sesquichloride; there are some patients who cannot take this, and then you may try some other preparation. You will, occasionally, meet with patients who are unable to take iron, in any of the ordinary forms, whilst they will bear it very well, and derive great benefit from it, in the shape of a chalybeate water. I attended some years ago a member of our profession who suffered from symptoms of fatty heart. He gradually became more or less breathless on exertion; he used to wake in the night with a sensation of suffocation; he suffered much from flatulence; and he complained of an occasional numbness at the extremities of the left fingers. He was pale, fat, and nearly 60 years of age. He had occasional palpitation of the heart, but the most distressing symptom he suffered from, and one which constantly drew his attention to his malady, was an irregularity of the heart's action. The heart would beat regularly for some minutes, and then there was a sudden "jerk," or "kick," as he called it. Sometimes, especially if the stomach were flatulent, the "kicks" or "jerks" of the heart were very frequent, and most distressing. I never detected any murmur over the heart; the sounds were faint, and the area of dulness normal. I advised the gentleman to curtail his labours, if he wished to prolong his life, and I put him on a course of iron. The iron he was unable to take, except for a few days at a time, as he suffered a good deal from dyspepsia. He tried various forms, but none of them agreed with him. He did not improve much under treatment, and, ultimately, with my advice, he gave up practice. Relieved, altogether, from the harass of practice, he began to improve. He subsequently went to Harrogate, and, whilst there, he drank the chalybeate waters. From these he derived great benefit, so much so, that the action of the heart became much more regular, and he gained much strength. He was still, however, obliged to lead a very careful life, and to be very cautious not to over-exert himself in any way. I mention this case as tending to show that, when the ordinary medicinal forms of iron do not agree, a chalybeate water may be beneficial.

You must avoid all depressing remedies in this affection; and although you may, at times, be obliged to give opium to relieve the distressing pain from which patients sometimes suffer, the less frequently you give it, I think, the better. In the later stages of the affection dropsical symptoms sometimes occur, and you may have to make choice of a remedy to endeavour to get rid of the dropsical effusions. Digitalis is occasionally given, and it is regarded by some physicians as a cardiac tonic. Of the value of digitalis in many cases of valvular disease, especially when there is hypertrophy of the heart, there can, I think, be no doubt; but it is not a remedy which, in my opinion, should be employed in cases of fatty heart. If you are ever disposed to give it, as a diuretic, from the failure of other remedies, I advise you to combine it with iron, but I think you ought to abstain from using it altogether.

CHAPTER IV.

CHRONIC DISEASE OF THE VALVES AND ORIFICES OF THE HEART — CARDIAC MURMURS.

(THE SUBSTANCE OF THREE CLINICAL LECTURES.)

GENTLEMEN,—I wish to call your attention to a class o cases, forming a considerable portion of those which ar to be found in our wards, and which you will frequentl be called upon to treat when you are engaged in practice I mean those cases in which there are persistent cardia murmurs, resulting from some diseased condition of th valves, or orifices of the heart.

I shall say nothing about the acute inflammations t which the lining membrane of the heart, and the valve themselves are liable, except in regard to the permanen mischief which these inflammatory attacks produce.

I have already, in some previous observations, pointe out to you the mechanism of cardiac murmurs, and have explained to you the general principles of physica examination, by which you can determine the site at whic the morbid sounds are produced, and the indications o structural disease which they afford.

By the observance of certain, for the most part, simpl rules, by listening over certain portions of the chest, and especially, by ascertaining the direction in which cardia murmurs are conveyed, you will, with a little experienc and with care, be able, *generally*, to predicate with grea accuracy the particular valve, or valves, in which the diseas is seated. I say, *generally*, for notwithstanding the perfection to which the diagnosis of cardiac valvular diseas has been brought, we meet with cases, at times, which are very puzzling, and about which we have, at first, some difficulty.

Let me say a word as to the import of a cardiac murmur. When, on listening over the heart, you hear a murmur, when you hear the healthy normal sound replaced by a blowing, or a whistling sound, you at once suspect disease of the valves, or orifices of the heart. Your thoughts do not go to the muscular fibre; there may be some diseased condition of the muscle, but the murmur directs your attention to the valves, and not to the muscle. But a cardiac murmur does not necessarily indicate organic disease; an altered condition of the blood, as in some cases of anæmia, gives rise to a murmur, which, as you have had frequent opportunities of observing, disappears under the influence of treatment. I cannot refer at length to these murmurs now, as my object is to deal with those which result from organic, or structural change.

You must not attach too much importance to a cardiac murmur. I mean, you must not take too unfavourable a view of the case of a patient, who comes to you with a murmur. The disease, of which a persistent cardiac murmur indicates the existence, is undoubtedly serious, and, from the secondary results which follow, it tends to destroy life; but these results are often very slow in coming, the distress, which the mere valvular disease gives rise to, is often very slight, and interferes very little with the ordinary duties of life. I could relate to you numerous instances of patients who have had valvular disease of the heart, and who have lived for many years, with very little discomfort. Much depends undoubtedly on the nature of the valvular affection, and the site of the disease. And let me here observe that you must not estimate the danger of a murmur in proportion to its loudness. The intensity of the murmur affords no indication whatever as to its character — serious, or otherwise. I direct your attention to this circumstance, as you might be apt to think that, because a murmur is not loud, therefore it is not important; and because it is loud, therefore it is very dangerous. Some of the loudest murmurs which I have heard have produced but few symptoms, and have gone on for a long time without interfering much with the comfort of the patients. On the other hand, it sometimes happens that the faintness of a murmur is an indication of danger; for it may result from a weakened, or degenerated condition of the muscular fibre.

You must not, as I have observed, take a desponding view of a case in which there exists a cardiac murmur, for you cannot judge, from the murmur alone, as to the exact amount of mechanical interference with the circulation which it produces, nor as to the rapidity with which secondary symptoms are likely to supervene. It is in these secondary symptoms, that — setting aside the liability to sudden death which always exists in these affections, but which, as I shall have to show you hereafter, is really very slight — the danger to your patients is to be found. And let me, at once, remind you that these secondary symptoms are, first of all, those which arise in consequence of the mechanical interference with the circulation of the blood; and, secondly, the effects which are produced on the muscular substance of the heart, not simply its hypertrophy, but the degeneration of the structure, and, consequently, the impairment of the function of the fibres themselves. In fact, the muscular fibres very frequently become the seat of fatty degeneration.

These valvular affections arise from several causes. You constantly hear me ask patients, in whom we discover cardiac murmurs, whether they have ever suffered from rheumatic fever; and you must have noticed how frequently the reply is in the negative. Acute rheumatism, attacking the endocardium and the valves of the heart, often gives rise to a permanent lesion of the valves. But this is, by no means, the most frequent cause of chronic valvular disease. We often find that our patients have only suffered from occasional rheumatic pains, and, therefore, if the valvular disease have been produced by rheumatism, it is by the chronic, and not the acute form. Again, there can be no doubt that gout is a cause of deposits about the valves; and in Bright's disease of the kidneys, changes of a similar character are very frequently met with. In emphysema of the lungs, also, not only do we find an alteration in the muscular substance of the heart, but valvular deposits are, by no means, infrequent. The valves are often, also, the seat of atheromatous deposits, and these undergo the, so-called, calcareous and ossific degenerations, and are the cause of cardiac murmurs.

I wish to impress upon your minds the fact, that most of these cardiac murmurs have a chronic origin, and that in only a minority of the instances, which we meet with, are they the result of acute inflammatory attacks. And there is yet another circumstance, which I wish, specially, to call your attention to, viz., that serious secondary symptoms are more apt to supervene rapidly, when the valvular mischief is the result of acute disease, than when it is of chronic origin. In the one case, the change in the structure of the valve has taken place rapidly, and the heart has not had much time to adapt itself to the altered mechanical conditions. Further, the inflammation may have, more or less, involved the muscular substance itself, which is, therefore, very liable to undergo fatty changes. But in the case of chronic disease, the deposit in the valves has taken place gradually — almost imperceptibly; the muscular substance has had time to accommodate itself to the altere conditions, and there has been no inflammatory attack t hasten the changes in the muscular fibre.

Either of the affections to which I have referred ma give rise to disease of any of the valves, or orifices of th heart, but those of the left side are, by far, the mos frequent seat of morbid changes. It is, indeed, very rar to meet with cardiac murmurs produced on the right sid of the heart, except where there is tricuspid regurgitation which frequently occurs in old-standing cases of dropsy and in emphysema of the lungs, when the right cavitie are much dilated. Practically, therefore, we may almos neglect the right side of the heart; and, in the remark which I am about to make, I shall confine myself to th consideration of those very frequent murmurs, which ar produced by disease of the left set of valves.

Let me then refer you, briefly, to the murmurs whic may be produced by some morbid condition of the valves or orifices of the left side of the heart. First, as regard their rhythm. A murmur is either systolic, or diastolic The systolic murmur is produced, either by some disease condition of the mitral valve, allowing of regurgitatio into the left auricle, or by some obstruction existing a the aortic orifice. There are two other conditions whic may give rise to a systolic murmur, which I will allud to presently. A diastolic murmur is caused, either b regurgitation of blood through the aortic valves,—a murmu of very frequent occurrence, or else by obstructive diseas at the mitral valve, which so constricts the orifice, tha a murmur is produced when the left auricle propels th blood into its ventricle.

The first point you have to decide, when you hear cardiac murmur, is, whether it is systolic, or diastolic, o whether it is, as we say, double, that is, both systolic and diastolic. Having settled this, you proceed to ascertain over what portion of the heart the murmur is best heard, and in what directions over the chest it is transmitted most distinctly; and you then draw your conclusions as to the particular set of valves which is the seat of disease.

Now with regard to systolic murmurs. I believe that a systolic murmur is sometimes produced without there being any regurgitation through the mitral valve, simply in consequence of a thickening of, or a deposit upon, the valve, which, giving rise to unequal vibrations, causes a murmur.

Cases are occasionally met with where there is a persistent mitral systolic murmur, and yet there are no indications whatever of regurgitation — no secondary effects either on the lungs, or on the general circulation. It is supposed, by some physicians, that a mitral murmur may be produced by the irregular action of the musculi papillares, allowing of slight regurgitation. It is quite possible that, when the heart is acting irregularly, an occasional murmur may be produced,—although I cannot say that I have heard one, under such circumstances; but, from what we know of the mode in which the mitral valve closes, it seems to me very difficult to understand how, during regular action of the ventricles, the musculi papillares can so act as to interfere with the closure of the valves.

Again, a systolic murmur may be produced by a roughened condition of the base of the aorta, from atheromatous or other deposit.

You will be surprised to find how little some people suffer from a valvular affection. It is really sometimes difficult to persuade them that they have a cardiac disease at all. They will tell you that they can do many things, which you think it is highly improper and injurious for

them to do, without feeling any ill effects. They can wal
well, they can go through a fair amount of manual labou
they can ride, perhaps even they hunt, and, except tha
they occasionally have palpitation of the heart, they com
plain of nothing. Very frequently, in these diseases, yo
will be consulted for some symptoms, rheumatic, dyspepti
or gouty, quite unconnected with the heart, and, on makin
an examination, you find that your patient has a cardia
murmur, with, perhaps, hypertrophy of the heart. It far mor
frequently happens that patients imagine they have seriou
heart disease, when there is nothing more than a functiona
disturbance, the result of disordered digestion, or of debility
than when they have actual organic disease. The palpita
tion and irregular action, which are so often met with a
functional disturbances, at once attract the attention of th
patient; whereas the gradual alteration of structure in th
organic disease gives rise, for a long time, perhaps, to n
symptom at all.

You probably recollect the case of James F., who wa
admitted under my care on the 18th of October last. Th
man was a sailor, and 23 years of age. He came her
in consequence of an attack of scurvy. He soon recovere
from the symptoms of scurvy, and he made no complain
of any other affection; but, about a week after his admis
sion, I examined his chest, and I then found evidence
heart disease. The following are the notes made at th
time of examination: — "The apex beats to the left of th
nipple, there is increased impulse of the heart, and increase
cardiac dulness. There is a loud double — systolic an
diastolic — murmur at the base, and at the second righ
costal cartilage, with a single systolic murmur under th
right clavicle. The heart's action is irregular; the puls
is 'jerking' and visible." The patient told us that h
had never suffered from rheumatic fever, that he felt n

pain, and that he had never noticed anything particular about his heart, except a little palpitation on going aloft, or on exerting himself more than usual. Thus you see that, in this instance, there had existed, at all events for a considerable time, perhaps for years, a serious valvular disease, leading to great enlargement of the heart; and yet the man was able to do his work as a sailor, and the only inconvenience he had suffered, was, occasional palpitation, a symptom which is felt on unusual exertion even when the heart is free from any disease. The small amount of suffering, and the few general symptoms which the disease produced in this case, are the points I wish to call your attention to.

When you have had some experience in auscultation of the heart, you will, I think, have little difficulty in making out the site of the different murmurs which you will hear; but there are some cases in which it is very difficult to form a definite opinion, when the patients are first seen. You may recollect the case of the boy, James R., who was admitted under my care on the 2nd of May last. He was 14 years of age, and he told us that he had never suffered from rheumatic fever, but that he had had rheumatic pains. When I first saw him, and indeed for some days after his admission, the action of the heart was so rapid and tumultuous, that I could not make up my mind as to the exact nature of the murmur. There was no doubt about the existence of hypertrophy of the heart, and of a double murmur, but I was unable to decide whether the murmur were, simply, ventricular-systolic, and ventricular-diastolic, or whether there was not, in addition, an auricular-systolic murmur. However, after the patient had been in the hospital a few days, and, especially, by taking the precaution to have him sent to bed, and kept in the recumbent posture for an hour or so before my visit, I was able to make out as follows: — Double murmur at the base, systolic and diastolic. The diastolic is prolonged, and leads up to the next systolic. Double murmur heard well at the second right costal cartilage, and under the clavicle; also at the lower end of the sternum, and to the right of the sternum. At the apex, there is a soft systolic murmur only; this is heard towards the left axilla. There is increased cardiac dulness. From this examination, I had no hesitation in concluding that there existed, in this boy, aortic obstruction and regurgitation, and, in addition, there was a strong probability of mitral regurgitation. I say strong probability, without speaking positively on the point, for this reason — there were none of the general symptoms of mitral regurgitation, and there was a possibility that the systolic murmur, heard at the apex and towards the axilla, might be transmitted from the aortic orifice; but at the same time I believe this was not the case, and that there was some regurgitation through the mitral valve.

Although it is very important, as a matter of diagnosis, and also, as I shall have to point out bye and bye, of prognosis, that you should be able to distinguish the sites of different murmurs, in the matter of treatment it becomes less important; for, whether the disease be at the mitral, or aortic orifice, you have to direct your attention to the secondary affections which ensue, and not to the diseased valves themselves, and the treatment must depend upon the nature of the symptoms which arise.

With reference to the question of diagnosis, let me call your attention to some cases which will serve to illustrate these cardiac murmurs; and to show you the unusual features which they, at times, assume.

You frequently find me directing your attention to a double murmur at the base of the heart, and in the direction of the right clavicle. The following case is interesting, as showing you the kind of disease which gives rise to such a murmur.

M. D. was admitted into the hospital in May, 1860. He was 23 years of age, and he told us that he had been well up to nine weeks before his admission, when he had rheumatism in the back. We found, on examination, a loud, rough, double murmur at the base of the heart, in the course of the aorta, and under the right clavicle. The second murmur was prolonged. At the apex of the heart the murmurs were faint, and in the left axilla, inaudible. The patient died suddenly, after remaining a few weeks in the house, and we made an examination of the body. The heart was large; the walls of the left ventricle were greatly thickened. The mitral valve showed a slight deposit, but there was nothing to interfere with its perfect closure. On the aortic valves two large, pendulous, firm, warty growths were formed. The aorta was atheromatous.

Here, then, was a condition of aortic valves which produced an obstruction to the passage of blood into the aorta, and gave rise to the systolic murmur; and, further, the valves were prevented from closing perfectly during ventricular diastole, and thus a diastolic murmur resulted. The murmurs were very loud in this case, and this is not the only instance in which I have found a very loud murmur produced by the existence of warty growths upon the aortic valves. The following is a case in point, and an interesting one for several reasons: —

Eliza McD., 25 years of age, a servant, was admitted into the hospital, under my care, on December 31st, 1860. She complained of having been ill for about five weeks, but it was very evident that the cardiac disease, under which she was suffering, had existed for a much longer period. On examining her chest, we found a loud systolic murmur, quite of a musical character. The murmur was loudest over the base, but it was also heard distinctly al over the chest, both in front and behind. Further, it wa heard when the stethoscope was placed on the abdomen, o on the arms ; and it was even audible, without the interventio of the stethoscope, when the ear was placed a few inche over the heart. Such were the characters of the murmur It was remarkable as being very loud, and very extensivel heard. It often happens that murmurs (especially aorti murmurs) are heard over a large portion of the chest, bu it is very rare that a murmur is conveyed along th extremities, and is heard on the abdomen. There is further, an interesting point about the murmur in this case viz., that it was heard when the ear was not in contact wit the body. In my own experience, I have only met wit this one instance, in which a murmur was thus audible.

The patient died on the 8th of February, after bein in the hospital about five weeks. We found, on examina tion, that the heart was very large, and the left ventricl much hypertrophied. The pulmonary and tricuspid valve were healthy. The mitral valve was slightly thickened, bu there was nothing to prevent its closure. The aortic valve had two warty growths upon them, about the size of smal peas. They were rough, and the valves themselves wer slightly thickened.

Here then, you see, we had a murmur with all the cha racters which are indicative of disease at the aortic orifice,— of aortic obstruction, in fact,—and we found, after death, tha such obstruction existed. There was no diastolic murmur the warty growths, unlike those of the last case I referre to, did not prevent the perfect closure of the valves.

You will meet with cases, which are, however, somewha rare, in which there is a single diastolic murmur hear at the base, and towards the lower end of the sternum and also, upwards, towards the right clavicle — a murmu depending on aortic regurgitation. When the murmur is heard alone, you can sometimes produce a systolic murmur by increasing the action of the heart, whilst, when the heart is acting quietly, the systolic murmur is not heard. I cannot find an instance of this kind amongst the cases which have been admitted in the hospital, and of which notes have been kept, but I have attended a gentleman, for some years past, who has this particular murmur very well marked. We can only account for the absence of the systolic murmur, by supposing that the ventricular surface of the valves is smooth, and that the deposit, which interferes with their perfect closure, has taken place on the upper surface.

Whilst I am speaking on this point, let me say a few words as to the fact which I have alluded to above, viz., the possibility of producing a murmur by exciting the action of the heart.

It occasionally happens, as in the case I have referred to, that, when the heart is acting quietly, there is no murmur with its systole, but when the action is increased, a murmur is audible. Again, it more frequently happens, that, on listening over the heart you hear a faint murmur, which, when the heart is excited, becomes louder, and well-marked. In examining a patient, if you have any doubt of the existence of a murmur, you should always take the precaution, before you give an opinion, to excite the action of the heart a little. If your patient be up, tell him to walk across the room, two or three times, and then listen to his heart; or if he be in bed, let him sit up and lie down, once or twice. This increased muscular exertion accelerates the circulation, and makes the heart contract more powerfully, and under these circumstances a faint murmur may become so intensified, that you can have no doubt about its nature.

Aortic regurgitation not only results from some disea condition of the aortic valves, but also in conseque of the aorta and the aortic orifice becoming dilated, with any enlargement of the valves taking place. The latt therefore, become incompetent to close the opening whi they arm, and, hence, there is regurgitation during ventri lar diastole. Under these circumstances a single diasto murmur is heard, provided there be no deposits in valves, and no roughened condition of the aorta. But more frequently happens that there is a systolic murm as well, produced either at the orifice, or else from diseased condition of the base of the aorta.

In such a case as I have described, you may occasiona find slight dulness at the upper part of the sternum, consequence of the enlargement of the aorta, and you m be led to suppose that an aneurism exists. Let me re you to the notes of the following case:—

William F. was admitted into the hospital, under my ca on the 7th of October, 1862. He had a double murm over the base of the heart. The systolic murmur was lou but the diastolic murmur, although prolonged, was not lou The patient told us that he had never suffered from rheuma fever, but had had some dropsical symptoms and dyspn for several months. There was evidence of hypertrophi heart, and the pulse was "jerking," although not ve strikingly so. We found slight dulness at the upper h of the sternum—at the spot where dulness very fr quently appears first in aortic aneurism—but we cou discover no pulsation. The man suffered during his st in the hospital from very distressing dyspnœa, more rese bling the form which is met with in fatty heart, a which I have already described, than the ordinary dyspno of valvular affections. The attacks were generally reliev by the administration of brandy. I have seen this for of dyspnœa in other cases of dilated aorta. There was a man in the hospital in March, 1863, who had a mass of schirrhous glands in the chest, and who also had this form of dyspnœa. The man died suddenly; and we found the aorta much dilated.

But to return to F.'s case. He left the hospital on January 17, 1863, and he died at his home, on the 5th of February, and through the kindness of the gentleman under whose care he had been, I had an opportunity of seeing the heart, and the aorta, after they were removed from the body. The preparation of them is now on the table. The heart was large, the left ventricle hypertrophied, and the aorta, from the commencement to the termination of the arch, dilated and atheromatous.

Such cases as those I have just referred to, where there is dilatation of the aortic orifice and of the aorta, with incompetency of the aortic valves, constitute a most distressing form of cardiac disease; I think I have seen more suffering from this condition than from any other form of heart affection — moreover, such cases are very liable to terminate suddenly.

Now let me call your attention to another murmur, about the frequency, if not the actual existence of which, there are differences of opinion. I mean the mitral diastolic murmur — a murmur produced by the passage of the blood, from the left auricle into the left ventricle, during the auricular contraction. Under ordinary circumstances, as you are aware, this portion of the rhythm of the heart is unaccompanied by sound; but when the mitral orifice is much contracted, it is quite conceivable that the auricular contraction may cause a murmur, which, of course, is diastolic as to time, or, as some prefer to say, presystolic, *i. e.*, it immediately precedes the ventricular contraction. My own opinion is that this murmur is not frequently heard, at least, I have not frequently heard it; but I have noted its existence, and I have little doubt that, in some cases, it is produced under certain conditions of the heart's action, whilst in other conditions it is absent.

There was a man in the hospital a few days ago in whom this murmur was well marked. I mean Joseph F., in L Ward. The patient applied here for a slight scalp wound, and it was discovered that he had a loud bruit over the heart. He was accordingly admitted under my care. He told us that, five years ago, he had rheumatic fever, and was ill for three months. He had also suffered from other attacks of rheumatism. We found, on examination, greatly increased dulness in the cardiac region, with increased impulse of the heart. At the base, there was a loud systolic murmur, the second sound being distinctly heard, although it seemed somewhat prolonged; but there was no diastolic murmur. Over the second right costal cartilage, and under the right clavicle, a single systolic murmur was heard. At the apex, and towards the left axilla, there was a loud prolonged systolic murmur, with a short diastolic murmur. The pulse was small and not "jerking."

In this case there was no source of fallacy as to the murmur, and however sceptical one might be as to the possibility of a mitral diastolic murmur being produced, I think an examination of the case would settle the doubt. When there is a double murmur, or even only a diastolic one, at the base, and a double murmur at the apex and towards the left axilla, although the second murmur heard at the apex and to the left *may be*, and probably often *is*, of mitral origin, still, such a conclusion is open to the objection that it may be the aortic diastolic murmur conveyed to the left; but, in the patient I am referring to, there was no aortic diastolic murmur, there were none of the characteristics of aortic regurgitation, the pulse was not "splashing" nor "jerking," and therefore the diastolic murmur must have been produced by the passage of the blood from the left auricle to the left ventricle; it was a short murmur, just as the auricular contraction is short.

But I must call your attention to some cases which presented the anatomical condition, viz., great constriction of the mitral orifice, which we believe is the cause of this murmur, but in which cases it was not heard.

Richard M. was admitted into the hospital, under my care, on the 1st of March, 1861. He complained of rheumatic pains in both legs, and of a pain over the heart. On auscultation, a systolic mitral murmur was heard. He died suddenly a few hours after admission.

On examining the body after death, we found the pericardium firmly adherent over the greater part of the heart, but especially anteriorly, where the adhesions were so strong, that, in order to separate them, it was necessary to use a scalpel. On laying open the heart, the aortic valves were found slightly atheromatous, but the chief seat of disease was the mitral valve, which, together with the chordæ tendineæ, was much thickened. The auriculo-ventricular orifice was so contracted as only to admit one finger. The right side of the heart was healthy.

Now, you will notice that the murmur in this case is put down as a systolic mitral murmur, and there are no notes made as to the exact site at which it was best heard. I only saw the patient once, and, thinking I should see him again, I did not dictate the particulars of the case to my clinical clerk at the time I made the examination. But I distinctly recollect that there was only one murmur heard—a systolic murmur — loudest at the apex and towards the left axilla. The patient died suddenly a few hours after my visit, and before any treatment had been adopted. We found evidence of old pericarditis; the pericardium was almost universally adherent; and there was a very contracted condition of the mitral valve. But there was no mitral diastolic murmur. Now let me call your attention to another case.

Catherine C., 30 years of age, was admitted, under my care, on August 30th, 1860. She had suffered from two attacks of rheumatic fever, and, when admitted, she was dropsical, and the urine was albuminous. She improved under treatment, and was discharged in November, but she was readmitted on the 23rd of the following January. Frequent auscultation revealed to us the following state of things: —

There was a systolic murmur, loudest at the apex of the heart and towards the left axilla, but heard also at the base, and along the course of the aorta. I have several notes to the effect that the murmur was single and systolic. There was, further, a distinct pulsation at the base of the neck, just above the sternum, which I believed to be due to a small aneurism, or a dilated aorta. The patient died on January 28th. The heart weighed twenty-four ounces. The cavities were dilated, and the walls thickened, especially the left. The mitral valve was thickened, and so contracted as only to admit one finger. The aortic valves were thickened by deposit. The aorta was dilated, and, at the commencement of the arch, it was about double its normal size. It was atheromatous.

Now, in this case, as in the last, there existed a very constricted mitral orifice, which admitted of regurgitation, and was of such a nature that, one would imagine, a murmur might have been produced by the rapid passage of the blood, from the auricle into the ventricle; but yet I never heard any other than the systolic murmur, and I listened frequently, not only during the latter residence of the patient in the hospital, but during her former stay, when we made out exactly the same auscultatory signs as afterwards.

Here are the notes of another case, bearing on this subject. Hugh D. was admitted into the hospital, under my care, on the 26th of August, 1861. He had suffered, he told us, from a severe rheumatic attack twelve years before admission. The patient was dropsical, and had many of the general symptoms of mitral regurgitant disease. He was in the wards nearly three weeks, and we established, on several occasions, the following facts: — There was a loud systolic murmur, heard best at the base, and towards the left axilla. The second sound was clear. The man died on September 14th. At the autopsy we found the heart much enlarged, and its cavities dilated. The valves on the right side were healthy. The mitral orifice was so contracted by hard atheromatous and bony deposits, as to admit only the tip of the little finger. The aortic valves were healthy. Here, there was extreme constriction of the mitral orifice, but there was no mitral diastolic murmur.

I think I have given you instances sufficient to prove that great constriction of the mitral orifice may exist, without there being any murmur produced by the passage of the blood from the auricle to the ventricle, and, therefore, that you must not look for a mitral diastolic murmur as a constant sign of obstructive mitral disease. My belief is that this murmur is far more frequently absent than present, even when there is great obstruction at the mitral orifice; and, further, that when you hear it one day in a case, you may not hear it the next. The presence, or absence of this murmur may possibly depend on the greater or less vigour with which the auricle contracts.

There was a man in the hospital a short time ago in whom I had some difficulty, at first, in deciding as to the nature of the murmurs which were audible. I mean John V., in

L ward. The patient had suffered for nearly three years, he told us, from palpitation, especially after exertion. He had œdema of the legs, and symptoms of œdema of the lungs, with great dyspnœa. We had evidence of great enlargement of the heart, and, on admission, and for a few subsequent days, the pulse was so quick, and the action of the heart so tumultuous, that I could not decide as to the exact nature of the murmurs which were audible. We could hear a double murmur, at the base, at the apex, in the axilla, and under the right clavicle. I had no doubt that there was aortic obstruction and regurgitation, with mitral regurgitation, but there seemed to me to be a mitral diastolic, as well as a mitral systolic murmur. After a few days' rest and treatment, the action of the heart became much more quiet, and we made out as follows: — "There is a double murmur, very loud at the base, at the second right costal cartilage, and all down the sternum — fainter at the apex, and in the left axilla. The systolic murmur is everywhere the louder."

The patient was greatly relieved from his symptoms, during his stay in the hospital, and he left us after remaining here a fortnight.

Now, my impression of this case is, first, that there was aortic obstruction and regurgitation. Of this I have no doubt, from the character of the murmur. Secondly, that there was mitral regurgitation. Of this I have no doubt; not only the general symptoms, but the character of the murmur at the apex, and towards the left axilla, indicated it. Thirdly, I think there was mitral obstruction, and that a murmur was produced at the period of auricular contraction. It is quite true that an aortic regurgitant murmur may be heard at times towards the left axilla, but when the patient was admitted I believe there was a mitral diastolic murmur, which disappeared as the action of the heart became more quiet. In connexion with this murmur let me read to you the notes of another case.

Thomas C., 43 years of age, was admitted into the hospital, under my care, on the 30th December, 1861. He had œdema of the legs, and had suffered, he told us, from rheumatic fever when he was about twenty-two years old. There was a loud, single, diastolic murmur at the base of the heart, and towards the right clavicle; and at the apex, and towards the left axilla, there was a double murmur. This is not a very frequent combination of murmurs. Although the second murmur, heard towards the axilla, was probably of mitral origin, yet, the objection which I have already referred to, the possibility of its being the aortic murmur transmitted to the left, might be brought forward against such conclusion.

Before I conclude my observations on these valvular diseases, let me refer you to the notes of a case in which a very unusual symptom was developed, about the nature of which I had no doubt at the time of its occurrence, but, unfortunately, I had no opportunity of verifying the diagnosis I had made.

Andrew G., 30 years of age, a sailor, was admitted into the hospital on the 14th of February, 1863. He told us that, eight years previously, he had suffered from rheumatic fever. On admission, he had œdema of the legs and face, which he said had existed for about two months. He had dyspnœa, a troublesome cough, and the urine contained a trace of albumen. There was increased cardiac dulness, and a double murmur was heard over the base of the heart, and towards the right clavicle. The murmur was scarcely audible at the apex. He remained without much change up to the 2nd of March, when we found him suffering from hemiplegia of the right side, and complaining of pain on the left side of the head. He was as well as usual when he went to sleep on the previous night, but on waking in the morning he found he could not move his right limbs. The paralysis of the arm was complete, but, when I saw him, he was able to move his leg slightly. There was no impairment of the mental faculties; he was perfectly conscious, and able to speak well; the face was not paralyzed, and there was no loss of sensation anywhere. He gained a little power over his leg, but there was no improvement in the condition of the arm. On the 17th of March, he was attacked with bronchitis, attended with profuse secretion, and on the 18th, he died. We were not able to make a *post-mortem* examination, but there can be little doubt that the case was one of cerebral embolism. In all probability, a portion of the deposits, about the aortic valves, had been displaced, and was carried by the circulation to some vessel in the brain. The occurrence of a case of this kind should always lead you to examine the heart in hemiplegia, in order to ascertain if there be any valvular disease, that you may clear up any doubt which may exist as to the paralysis being the result of an embolus detached from the valves.

According to my experience, the aortic valves are the most frequent seat of the disease in these valvular affections. Taking 68 consecutive cases, of which I have notes, I find that in 32 there was disease of the aortic valves, in 22 disease of the mitral valve, and in 14 disease of both valves. Of 79 cases collected by Dr. Barclay, from *post-mortem* records, the proportion in which the different valves were diseased was as follows:—In 36 cases, both valves were diseased, or 45 per cent.; in 26 cases, the aortic valves were diseased, or 33 per cent.; in 17 cases, the mitral valve was diseased, or 21 per cent. And the same proportion, as nearly as possible, is maintained in a larger series of nearly 200 cases. Now these statistics show that, in a very large proportion of cases where death takes place as a consequence of valvular disease, both the mitral and aortic valves are affected; but I believe it will be found that in many of such cases, whether of acute or chronic origin, the aortic valves are the first to become involved, and that the mitral disease is secondary.

I now pass on to say a few words of the treatment of valvular diseases of the heart, and I must confine my remarks to the general management of these affections, without attempting to give you directions as to the treatment of particular symptoms. In watching the cases which you see admitted into the hospital, you cannot fail to be struck with the difference, as regards symptoms, which different patients present. Some, indeed, seem to suffer very little, whilst in others the distress produced is very great; and this difference does not depend so much on the site of the valvular disease, as on other circumstances.

Let me impress on you one very important fact in connexion with these maladies, viz., that from whatever cause they arise, when once they have become chronic, we possess no means of removing them. Issues, setons, etc., over the heart, are sometimes used, but except for the relief of pain, when they may occasionally do good, they are, I think, valueless. It is very important that you should recognize the cause of the valvular disease, so that if it be, for instance, gout, or rheumatism, you may adopt such a course of treatment for your patient, as may best tend to keep the rheumatic or gouty symptoms in check, and so prevent, as far as possible, any further deposit on the valves. And in this respect I believe much can be done. But, in addition to your endeavours to correct that state of the system which has led to the valvular disease, you must direct your efforts to check the development of the secondary changes which it tends to produce. You know what those changes are—hypertrophy of the heart, with dilatation of its cavities;

interference with the circulation, pulmonic and systemic; dropsy; pulmonary apoplexy, etc.

As I cannot dwell long on the subject of treatment, let me impress on you, in as few words as possible, what I consider the most important points to be aimed at. When there is a mechanical interference with the passage of the blood through the heart, hypertrophy of the latter necessarily results, and it is a salutary process. If you were to check it you would do harm. You should endeavour to keep up the vigour of the heart, and to prevent its structure becoming weakened, or degenerated. Dropsy, and other secondary results of valvular disease, are much more likely to supervene, if the heart become weakened and dilated, than if its fibres be kept in a healthy condition. It is remarkable how long people will live, even when they are undergoing severe manual labour, with a valvular affection, provided the muscular substance undergo no degeneration. The great danger consists, in my opinion, in the weakening of the muscular fibre, and, especially, in its fatty degeneration. I have never examined the heart of a person who had died suddenly with valvular disease, without finding the muscular fibres in an advanced condition of fatty degeneration. And in cases where death has been produced by dropsy, or some other secondary affection, and when the valvular disease has been long-standing, I have generally, if not universally, found a similar state of the muscular fibres. You, by no means infrequently, see cases in which valvular disease has existed for many years, and yet the patients have perhaps been living a life of hard manual labour. In these cases the heart becomes enormously hypertrophied, and there may be few, or no dropsical symptoms until after the lapse of a very long period. I believe this result must be attributed to the fact that, although the habits of life of the patient have, on the one hand, been injurious, and have led to great hypertrophy of the heart, they have yet prevented the degeneration of the muscular fibre, and thus the injury has been, to a certain extent, counterbalanced. On the other hand, we constantly see persons of sedentary habits, with a flabby muscular system, rapidly succumb to the secondary results of valvular disease. These are important points for you to bear in mind in reference to the prolongation of life in these affections.

Setting aside, for the present, the secondary symptoms which we so often see in the patients who are admitted into our wards, let me say a few words as to the management of valvular disease, in which such symptoms have not developed themselves, or have been relieved.

I think it is not desirable to insist too rigidly upon the abstinence from exercise in these cases; on the contrary, it is, in my opinion, very important that a moderate amount of exercise should be taken. It is, undoubtedly, desirable to prevent all severe exertion, all hard manual labour, and all violent exercises; but moderate exercise is not to be forbidden. You should recommend your patients to live in a dry, bracing atmosphere, if possible, and to be much in the open air. The rules which I have laid down on these points, in speaking of fatty disease of the heart, are applicable to valvular affections. I would warn you against allowing your patients one kind of exercise, viz., horse exercise. Although people with heart affections frequently do ride, and sometimes even hunt, I think it is for them a dangerous pastime, unless they never put their steeds beyond a foot's pace. No such objection, as applies to riding, applies to driving, yachting, etc., etc., and from these exercises patients derive great benefit.

In the regulation of the diet it is only necessary to observe ordinary rules. You must bear in mind that all disorders of digestion, flatulence, etc., are apt to produce palpitation and irregular action of the heart, in these affections, and are therefore to be specially guarded against. The diet should be nutritious and not bulky, and should include a moderate amount of stimulants. Your object should be not to overload the blood-vessels, or the circulation may be embarrassed. You should be very careful how you suddenly change the habits of life of a person who is suffering from a heart affection. Whatever change you wish adopted — either of diet, stimulants, or exercise — should, unless under special circumstances, be accomplished gradually. There is, in all sudden changes, a danger of sudden death.

You may prescribe any of the ordinary tonics for your patients, as they may be required for any particular symptom, but there is one medicinal substance, of the great value of which in cases of heart disease there can, I think, be no doubt — I mean, iron. I have already spoken of its use in fatty heart, and the principles for its administration, which I then gave you, will apply to its exhibition in valvular disease. You see me constantly give it; and my belief is that we possess, in it, an agent which, in connexion with other measures such as I have referred to, is capable, when given in properly regulated doses, and continued for a long time, of arresting the degenerative changes which so constantly supervene in these affections. The form I most frequently use is the tincture of the sesquichloride, and I know of no better preparation. It should not be given in large doses, and it should be persevered with, not only for weeks, but for months and years; in fact, unless there are contra-indications, from the presence of gout or rheumatism, or of some other symptoms, it should be taken regularly, with only occasional intermissions.

You will be called upon to treat the palpitation which comes on in these valvular affections. You frequently see cases which have been admitted into the wards on account of this symptom. Very often, rest in bed, with the application of belladonna lotion, or a belladonna plaster, over the heart, or even rest alone, soon causes the palpitation to cease; but you sometimes see me give digitalis when the case is severe. However, I generally omit it soon, and I then prescribe iron; and in many cases of palpitation I prescribe digitalis and iron together; and I think this is a very good plan, except when the palpitation is extreme, and then I have found the digitalis, given alone, act best. Palpitation of the heart, coming on without the presence of organic disease, generally indicates a depressed state of the system, or some disorder of the digestive organs. It comes on under conditions of great nervous exhaustion, and as the result of wasting discharges from various causes, and it is to be relieved essentially by stimulants and tonics. And the same remarks apply to the palpitation of valvular diseases. It is often brought on by causes which have affected the general health, and is best relieved by a stimulating and tonic treatment. You must not, however, adopt any routine practice in such cases, but you must be guided by the circumstances of each individual case.

Many of the patients who are admitted with valvular disease are suffering, at the time, from dropsical symptoms, more or less marked. When the dropsy is only slight, it is often got rid of rapidly, without the use of any diuretics or purgatives, by simply giving iron; but when it is more extensive something further is required, and then you will find digitalis of great value.

But, in treating these cases of extensive dropsical effusion, you must bear in mind that it is not simply sufficient to get rid of the accumulations. They will very soon recur unless you can improve the condition of the heart, and of the general circulation. Whilst, therefore, you give digitalis, or some other diuretic, you must also give iron, and this latter must be continued after the effusions have disappeared. Let me warn you against the practice of giving powerful purgatives to patients with heart disease. You may possibly by such means get rid of a large quantity of fluid, but the result may be accomplished at the expense of much of your patient's strength, and possibly at the risk of his life. You must never forget the effect which depressants have on the circulation, and the danger of fatal syncope, which always attaches to heart disease.

I think you should avoid giving opium in diseases of the heart, unless you are obliged to have recourse to it under special circumstances. It has a tendency to lower the circulation, and, therefore, is objectionable.

Before I conclude, let me say a few words as to the use of the sphygmograph in diseases of the heart. You frequently observe that I have the tracings of the pulse taken in cases of valvular affection, but I cannot say that I derive much use from it in arriving at a diagnosis. The fact is that I have generally made up my mind as to the nature of the case, before the sphygmograph is used, from the murmurs which I hear; and although the pulse markings are characteristic of certain valvular lesions, they do not afford so ready and certain a means of diagnosis as auscultation. At the same time, I think it highly probable that, with more prolonged and extended use, the instrument may become valuable in reference to certain doubtful cases.

There is another class of cases — cases of acute disease, in which it is thought that this instrument may eventually become of great value — not as a means of diagnosis, but with reference to prognosis, and the mode of treatment that should be adopted. I have had no experience of the use of the instrument in this respect, and therefore I can express no positive opinion of its value.

CHAPTER V.

ON SUDDEN DEATH IN RELATION TO HEART DISEASE — AND ON THE PROBABILITIES OF LIFE IN VALVULAR DISEASE OF THE HEART.

THE disease of the heart which is most liable to terminate in sudden death is, undoubtedly, fatty degeneration of its muscular fibre. It is unnecessary to refer to statistics to prove this statement: the experience of most practitioners of medicine will confirm it. When fatty disease attacks the muscular substance of the heart, there is a gradual obliteration of its contractile element, and a gradual diminution of its contractile power. To such an extent does this sometimes take place, that, on making an examination of the tissue of the heart, we find but little evidence of its muscular nature; and we are surprised, not that death has occurred, but that life has been so prolonged. Death, not unfrequently, takes place suddenly in this disease, without the previous occurrence of any well-marked symptom, such as to arrest the attention of the patient, and warn him of his dangerous malady; and although it is probable that, in all such cases, a careful examination would reveal evidence of an enfeebled heart, or of certain reflex phenomena, slight, but important in a diagnostic point of view, yet, the absence of prominent features — such, for instance, as usually characterize the progress of valvular disease of the heart, and of certain affections of its muscular walls — often leads the sufferer to imagine that no serious malady exists. And it is a circumstance of no little interest and importance, that, even when fatty degeneration of the heart is far advanced, we occasionally find the pulse moderately full. I have known instances where this condition of the pulse has misled as to the nature of the disease.

But beyond this question of the great liability of fatty disease of the heart to terminate in sudden death, there are some further points, in relation to sudden death from valvular disease, of great practical importance. Amongst these are — 1st, *the probabilities of sudden death in valvular disease;* and, 2nd, *the particular form of valvular disease most liable to terminate in sudden death.*

In regard to the first point, I think it may be safely affirmed that, speaking generally, the proportion of cases of valvular disease, terminating in sudden death, is very small. In the large majority of cases death results slowly, from the secondary consequences of the affection, dropsy, or some other diseased condition. Dr. Barclay has recorded a series of seventy-nine fatal cases of valvular disease which occurred in St. George's Hospital. Two only of the deaths are mentioned as having been sudden. This proportion of sudden deaths is very small, and, perhaps, must not be taken as the usual one. My own experience certainly gives a larger proportion.

In relation to the second point, the particular form of valvular disease most likely to terminate in sudden death, I am not aware that any statistical tables exist which would tend to determine the question. It would be a matter of much importance, considering the strong impression, which prevails amongst the public, of the great liability to sudden death in heart disease, if we could arrive at any precise conclusions in regard to this subject — if, in fact, we could, by the examination of a large number of cases, deduce a rule of probability applicable to these valvular diseases. The experience of a single individual, unless extraordinarily large, would scarcely serve for any definite conclusions; but if a number of practitioners were to direct their attention to this particular point, the most valuable statistics might be obtained. Dr. Walshe, in the last edition of his work on "Diseases of the Heart," has entered somewhat into this question, and he states that, according to his experience, the form of valvular disease most liable to terminate in sudden death, is, uncomplicated aortic regurgitation. On the other hand, Dr. Stokes is of opinion that mitral regurgitant disease is most liable to fatal syncope.

Theoretical considerations lead me to the conclusion that, of the two forms of disease just mentioned, mitral regurgitation is more liable to terminate in sudden death than aortic regurgitation. In the latter affection the dilatation and hypertrophy of the left ventricle are especially salutary. If the disease is chronic, the ventricle gradually adapts itself to its altered requirements, and, for a time, but few symptoms, sufficiently severe to attract the attention of the patient, may result. In consequence of its dilatation and hypertrophy, the ventricle is able both to hold a larger quantity of blood than in health, and to contract with greater power, so as to throw all its contents into the aorta. The arterial tubes thus become well filled by each ventricular systole — in fact, they receive more blood than when the heart is in a normal condition; but, in consequence of the imperfect closure of the semilunar valves, they lose a portion of this blood, and hence the sudden collapse of the arteries after their diastole, which gives so peculiar a character to the pulse in aortic regurgitant disease. Now, as the structures of the body get well supplied with blood *so long as the ventricle retains its vigour*, there is, speaking comparatively, but little element of syncope, and sudden death. On the other hand, when the mitral valve is diseased, so as to allow of regurgitation, a portion of the blood, which ought to go to fill the arteries, passes back into the left auricle. Hence these cases are characterized by a small pulse — a a pulse of little volume. If the amount of regurgitation be large, the quantity of blood passing into the systemic vessels is, necessarily, small. Here we have the element of syncope; and, as the result of an unusually feeble contraction of the ventricle, or of some embarrassment to its action, sudden death may ensue. Whether such embarrassment is more likely to take place in mitral, than in aortic regurgitation is a subject for careful study and observation.

On looking over the cases of sudden death amongst my own patients, I find that I have had about an equal proportion of deaths from the two forms of disease to which I have referred, and, consequently, my own experience tends neither to confirm, nor to contradict the opinion I have expressed on theoretical grounds. The question is one especially for statistics, and it is chiefly with the view of eliciting facts, and the opinions of those who have had a larger experience than I have, that I have brought the the subject forward.

My own belief is, that it is a very rare thing for valvular disease to produce sudden death, unless the muscular substance of the heart has undergone a weakening, or a degeneration of its fibre. As long as the muscle retains its vigour, the grand cause of syncope is wanting. In all cases of sudden death from heart disease in which I have made a *post-mortem* examination, I have found fatty degeneration of the muscular fibre to a greater or less extent. The recognition of this fact is of much importance, as it points out the line of practice that should be adopted in the management of all cases of valvular disease.

With regard to the probabilities of life in valvular disease, I believe, that the balance is in favour of aortic, rather than of mitral disease. In the former, it is often a long time before severe secondary symptoms result, and, in fact, until the ventricle becomes much dilated, and the mitral valve incompetent, the circulation is very often well maintained. In mitral disease, however, there is at once an impediment produced to the pulmonary circulation, which may rapidly lead to serious secondary symptoms. It is quite true that we see life greatly prolonged in mitral disease, as well as in aortic disease, but this does not affect the general question.

CHAPTER VI.

THORACIC ANEURISM.

There are few diseases more interesting to the practical physician than thoracic aneurisms. The insidious character of their origin, the frequent obscurity of their symptoms, the occasional rapidity of their progress, and their tendency to a fatal issue, render them a study of more than ordinary importance. That their diagnosis at an early stage, and the discrimination of them from other forms of thoracic tumours, as well as from certain affections of the heart, are often attended with great difficulty, no one will deny. It is, however, in the direction of early diagnosis that our efforts should especially be made, for on this, the value of our treatment, and our hope of prolonging life, must mainly depend.

Without attempting to give a complete history of thoracic aneurism, I propose to refer to some of the cases which have been under my care, as they will serve to illustrate the symptoms which characterize the disease, its progress, and method of termination, as well as the more or less permanent relief which sometimes occurs in it from the use of remedial measures.

Case I.—*Aneurism of the Arch of the Aorta. Sudden Death from Rupture of the Aneurism into the Left Lung.*—William T., forty-eight years of age, a sailor, was admitted into the Liverpool Northern Hospital, under my care, on the 6th of May, 1861. He gave the following history:—He had followed his occupation on board ship up to ten days before his admission. For about a week he had suffered from a cough, which was unattended with expectoration. For two or three nights he had been unable to lie down, and the day before he came to the Hospital he had a fit of dyspnœa which lasted for about half an hour. He said he had never felt any throbbing in his chest, nor any other unpleasant symptoms except those just mentioned. I saw him for the first time on the 8th of May, and just as I was about to make a physical examination of the chest, he was suddenly seized with a violent attack of dyspnœa. The paroxysm resembled in every respect a severe asthmatic seizure. The surface of the body became livid, and was covered with a profuse, clammy perspiration. These symptoms lasted for nearly a quarter of an hour, when chloroform was cautiously administered by inhalation. After a short time this gave relief, the spasm passed off, and the breathing became natural again.

At a subsequent visit, I made a careful examination of the patient, under the impression that there must be some thoracic tumour. He had had a slight return of the dyspnœa, and had found relief from smoking stramonium. He complained of a feeling of weight in the chest, opposite the upper part of the sternum, and had a constant ringing cough.

There was the usual amount of dulness on percussion in the cardiac region, with dulness opposite the upper half of the sternum, and in the first, second, and third intercostal spaces, for about two and a half inches to the left of the median line. A distinct impulse was felt in the second and third left intercostal spaces. The heart-sounds were normal, and no bruit was audible over the seat of impulse and dulness. The right brachial artery was hard, and felt under the finger like a firm cord. No difference was noted in the pulse of the two sides.

On the 20th of May, a fortnight after admission, I made the following note: — "He appears to be improving, and complains of no pain, nor of any unusual feeling about the chest, but he has dyspnœa on exertion. He is unable to lie down, and uses a bed-rest." On the 28th he had so far improved as to be able to lie down without the bed-rest. He continued without much change, either in the general symptoms or physical signs, up to the 8th of June, when he had slight hæmoptysis. On the 17th, the hæmoptysis returned to a greater extent, and on the 20th, at one o'clock a.m. the house-surgeon was called to him, and found him dead. He had suddenly thrown up a large quantity of blood, and died immediately.

A *post-mortem* examination was made the following day. On opening the thorax, the cause of the dulness and pulsation was apparent: a tumour occupied the situation of the dulness, and, on examination, it was found to be an aneurism connected with the ascending portion of the arch of the aorta. The tumour was about the size of an orange, and had pushed its way upwards, and to the left, into the apex of the left lung, in which it was, in part, embedded. The aneurism was of the dissecting kind; it had separated a portion, about an inch and a half in length, of the inner coats of the artery from the external coat, which latter formed the covering of the aneurism. The cavity of the aneurism had some firm fibrinous layers in it, and very little soft clot. An opening was found in its upper part, which led into the substance of the left lung. The left lung was full of blood; the right lung also contained blood. The heart was very pale and soft, apparently fatty; it was not examined under the microscope.

Remarks.—The most important points for reference in this case are, 1st, the insidious manner in which the disease crept on, and the proportions it assumed, before it gave rise to symptoms which attracted the attention of the patient. That it had existed for a longer period than ten days before the patient applied for admission into the hospital, there can be no doubt, and in all probability the tumour had developed itself to a considerable extent, and was making its way into the substance of the left lung, when the first paroxysm of dyspnœa occurred.

2nd. I would refer to the character of the first important symptom — viz., the asthmatic seizure — a reflex spasm of the whole of the bronchial tubes, set up, no doubt, by the pressure of the tumour on the pneumogastric nerve.

Lastly, I would allude to the occurrence of hæmoptysis on two occasions preceding the fatal one. This is, almost always, an alarming symptom, and if, when there is a supposition of aneurism, it occur in more than a very small degree, it should put us on our guard as to the probability of an early fatal issue. It is quite true, however, that in some cases of aneurism, hæmorrhage, even of a profuse kind, has taken place some weeks, and even months, before death has occurred. In such cases, the opening which has been made has been plugged up by laminæ of fibrine, or coagulated blood.

Case II.—*Aneurism of the Arch of the Aorta. Rupture into the Trachea. Sudden Death from Hæmorrhage.*—Philip R., 38 years of age, a Frenchman, by occupation a sailor, was admitted into the Liverpool Northern Hospital, under my care, on the 22nd December, 1861. He gave the following history:—His health had been good, he said, up to the previous July, viz., about six months before admission. On the 4th of that month he left this country for St. Louis. Whilst at sea he had dyspnœa, and pain in the chest. When he reached a warmer climate his symptoms were greatly relieved; in fact, he felt but little of his ailment. After staying at St. Louis for eight weeks, he returned home, and as he approached this country his symptoms reappeared. He reached Liverpool on the 12th November, and a week afterwards he went into hospital for two days, when, feeling much better, he left. From the time he went out to the date of his re-admission, his symptoms continued with more, or less severity, and he was unable to resume his work.

When first seen by me, on his second admission, he was suffering from many of the symptoms of laryngitis. There was a hoarse cough, with somewhat stridulous breathing; harsh laryngeal sounds were heard on auscultation, with pain on pressure over the larynx. There was a history of an old syphilitic affection. The chest was examined, but not carefully, and no dulness was detected. The breath-sounds over the lungs, although feeble, were good. The patient was unable to lie down, and was propped up by a bed-chair.

The symptoms continued with but little change; the face assumed an aspect of great anxiety; there was urgent dyspnœa, with loud, ringing cough. At times, however, the breathing was natural.

On the 3rd January, I made a very careful examination of the patient. I had previously expressed an opinion that there was some lurking disease, probably aneurismal, and that the laryngeal affection was of a spasmodic character, the result of some distant irritation.

On feeling the pulse of the two arms, a marked difference was perceptible. On the right side the pulse was very small, on the left moderately full. There was slight dulness opposite the first piece of the sternum, extending two inches downwards from its upper edge, and one inch and a half to the right of the bone. There was an indistinct pulsation in the first and second right intercostal spaces, close to the sternum, and a very slight beating was observable, on close observation, at the lower part of the neck, on the right side. There was a slight prominence of the right side of the chest at the seat of dulness. The heart-sounds were normal, but faint; no bruit was audible over the seat of pulsation, or elsewhere.

On the 4th of January he complained of pain in the right arm, and in the right side of the chest, and of dyspnœa after swallowing. On the night of the 6th January he had a most violent attack of dyspnœa, which lasted for two hours. On the 9th, I made the following note:—"Pulse 76, regular; very small in the right wrist; there is more dyspnœa." On the 14th, the symptoms were more urgent; there was more cough, and some expectoration. On the 18th, the pulse was intermittent; the sputa were purulent and copious. Dysphagia was complained of.

But little change took place from this period, except that indications of the existence of a low form of pneumonia became daily more marked. The paroxysms of dyspnœa, however, were less frequent. He was quite unable to lie down, or even to lean back. On the morning of the 31st January he died. He had got out of bed, when he suddenly coughed up a large quantity of blood, and before the house-surgeon could reach him he was dead.

Autopsy.— On removing the sternum and costal cartilages, a tumour was seen occupying a position behind the upper half of the sternum, and extending slightly on either side. This tumour was an aneurism, about the size of an orange, connected with the ascending portion of the arch of the aorta. Anteriorly, the aneurism had pressed against the upper part of the sternum, and had produced slight absorption of that bone. The tumour contained, in front, a quantity of soft fibrinous clot, evidently of not very old formation. The tumour pressed on the trachea, and had ulcerated through that tube, at about an inch and a half above its bifurcation. The aneurism had thus burst into the trachea. The opening, as seen at the autopsy, was plugged with a soft, dark-coloured coagulum.

The anterior portions of the upper, middle, and lower lobes of the right lung, were the seat of grey hepatization. Both lungs contained blood.

The heart was rather small; it had a deposit of fat on its surface; its valves were healthy. The aorta was atheromatous; the larynx was healthy.

Remarks.—The laryngeal symptoms and the supervention of pneumonia are important features in this case. It is no uncommon thing to find that the first symptoms to attract attention in thoracic aneurism are those of spurious laryngitis; and the symptoms are of so deceitful a character that even the most cautious physicians have sometimes been misled by them, and have recommended that the operation of tracheotomy should be performed, under the impression that there has been some organic constriction of the glottis. In the case which I have just related, I did not satisfy myself for a few days of its exact nature, but the careful physical examination I made on the 3rd of January at once removed all doubt from my mind.

There was once circumstance which, on a mere superficial consideration, would tend to produce an erroneous impression. It was this: —On first seeing the patient I noted the larnygeal symptoms alone, and I directed that hot moist flannels should be kept constantly applied over the larnyx. From this the patient experienced great relief, not only at first, but up to the period of his death; so that, whenever he found an attack of dyspnœa coming on, he used to ask the nurse to apply the hot flannels. It is somewhat curious that a local application of this kind should give relief to a spasm depending upon a distant irritation.

The important practical point to be drawn from the case is, that whenever laryngeal symptoms of such a nature occur, we should look well, in order to ascertain whether they may not be simply, and solely, of a reflex character, before we give a decided opinion of their nature, or before we recommend that the trachea should be opened.

I would remark next with reference to the inflammation of the right lung. This is a result which very frequently occurs in thoracic aneurism, and seems to be due to the pressure which the tumour exercises on the nerves supplying the lungs. The result resembles that which follows when the pneumogastric nerves are divided; for we then observe, first, a passive congestion, and, subsequently, a low form of pneumonia, developed. The symptom is one of very considerable interest and practical importance, as it may be the means of producing a fatal result before the aneurism has had time to ulcerate into any great cavity, so as to give rise to death from hæmorrhage.

CASE. III.—*Thoracic Aneurism? Improvement under Treatment.*—William R., sixty-three years of age, a labourer in an iron-foundry, was admitted into the Liverpool Northern Hospital, under my care, on the 25th August, 1862.

The history he gave was as follows:

He had been ill about three weeks, but had only left off work for ten days. The first symptoms he felt were "a sort of fainting when he stooped, and a choking sensation in the throat." During the previous week he had occasional fits of dyspnœa. He said he had never had any serious illness; he had suffered slightly from rheumatism, but never from rheumatic fever.

He complained of a sensation of weight in the chest and of pain in the neck; the face was swollen and livid. This swelling had existed, he said, for about a week. The superficial veins of the chest and neck were enlarged, and formed a visible plexus. The veins of both arms were very tortuous. The pulse was 76, and much smaller in the right arm than in the left. He breathed, easily, when he sat or stood, but, with difficulty, when he lay down. In the latter position the breathing became of a stridulous character. There was œdema of the integuments of the chest, but not of the legs.

The chest in front was resonant all over the left side. On the right side there was resonance below, and at the upper and outer part. There was dulness over a space occupying the upper half of the sternum, and extending two and a half inches to the right of that bone, but not passing to its left. Both lungs were resonant behind. There was slight prominence of the right side of the chest, opposite the seat of dulness. No pulsation could be felt over the tumour; the cardiac sounds were audible over it, but there was no bruit. The apex of the heart was felt, and seen, beating two inches to the left of, and three and a half inches below, the left nipple. A systolic bruit was heard at the apex of the heart. There was increased dulness in the cardiac region. Both pupils were of the same size. The urine was examined and found free from albumen.

On the 29th, there was more swelling of the chest walls, and the upper extremities were also swollen, especially the right. On the 2nd September the dyspnœa was rather less. There was distinct pulsation over the tumour. On the 9th there was less venous congestion of the surface of the chest, and the breathing was less stridulous when he lay down. He was better able to lie on his back. The cardiac systolic bruit was audible at the base, as well as at the apex of the heart.

On the 16th, the right pupil was observed to be more contracted than the left.

On the 4th October, I made the following note—"He fell down last night, but soon recovered himself. There is great œdema of the right arm. The extent of dulness is somewhat greater than it was. Both pupils are of the same size.

On the 25th, the dulness was found to extend three inches to the right, and an inch and a half to the left, of the upper half of the sternum. The impulse was about the same ; the breathing was decidedly better.

On the 11th November he spat up a little blood. He complained of feeling a weight under the right breast. The tumour was more prominent, but the impulse about the same. Pulsation could be felt deep in the episternal notch.

At the end of November, the œdema of the arms, and the venous congestion of the chest walls, had almost subsided. On the 20th December the dulness had increased; in fact, it extended nearly over the whole of the anterior part of the right side of the thorax. There was less pulsation. No breath-sounds could be heard in front on the right side, but vesicular breathing was audible behind.

The patient remained in the hospital till the beginning of January, 1863, when he was discharged. There was, at that time, no œdema of either arm, and no venous congestion of the walls of the chest. The pulsation over the tumour was decidedly less than it had been. He could lie down, slept well, and had very little dyspnœa. He was able to go about, and, as long as he kept himself tolerably quiet, all went well.

Remarks.—Although there was no opportunity, by *post-mortem* examination, of verifying the diagnosis in this case, there can be, I think, little doubt of the nature of the disease, which must have been either aneurism or pulsating vascular tumour. The symptoms and progress of the case point to the former affection. The chief interest lies in the steady and progressive improvement which took place, as contradistinguished from the progressive decline, and rapidly fatal issue which characterized the two preceding cases. That the tumour became larger and more solid is evident from the increase in the extent of dulness, and the diminution in the impulse. There can be no doubt, I think, that a deposit of fibrine took place in the aneurismal sac, and that its cavity became diminished in size; that, in fact, the disease was in process of cure, so far as cure can be accomplished in such cases.

The treatment adopted consisted in occasional cupping, to the extent of four or six ounces, the careful regulation of the diet and of the amount of exercise taken, and the administration of a purgative from time to time. The patient was cupped about once a month. He took digitalis for a short time. The bowels were kept well open, but he was not purged. His diet consisted of a small quantity of meat daily, and he was ordered to take very little fluid and vegetables. He was, except at first, when he was kept quite quiet, allowed to go about, and was a good deal in the open air.

Case IV.—*Aneurism of the Arch of the Aorta. Death from Secondary Diseases. Autopsy.*—Celia P., 30 years of age, a somewhat plethoric woman, was admitted into the Liverpool Northern Hospital, under my care, on the 10th April, 1862.

She gave the following history: In November, 1861, she first felt a beating over a small spot opposite about the third, or fourth left costal cartilage. The beating gradually increased, and a swelling soon became apparent; this latter also increased, but for the nine or ten weeks previous to her admission, she thought there had been no enlargement in it. She had suffered a good deal from pain in the back, and left arm. She complained of a constant beating in the chest, and of inability to lie on the left side; there was pain in the left shoulder, and pain shooting down the left arm, with numbness and tingling of that limb; the veins of the left breast were enlarged. There was a distinct bulging of the left side of the chest, extending from about the level of the lower third of the sternum nearly up to the clavicle. The swelling was seen, and felt, to pulsate strongly. The pulsation was strongest, and the swelling most prominent, opposite the fourth left rib. There was dulness on percussion over the swelling; this dulness reached from the cardiac region up to the clavicle, and extended three inches to the left of the sternum. The pulse was 92, and regular; much smaller on the left side, both in the arm and wrist, than on the right. There was a slight bruit with the first sound of the heart. There was very little dyspnœa, and the patient could lie on her back; the chest was resonant behind, and the breath-sounds were normal; there was a good deal of pain over the tumour.

The patient remained in the hospital till the 10th May, when she wished to go home to her friends. At the time of her discharge the pain over the tumour was much diminished, and, generally, she was improved, but there was no diminution in the size, and impulse, of the tumour. She suffered whilst in the hospital but little from her ailment, and could scarcely be made to believe that she was the subject of a serious malady.

The treatment adopted consisted in the application of a belladonna lotion over the tumour—which had a decided effect in relieving the pain—rest, a somewhat spare diet, an occasional aperient, morphia at night, and one venesection to six ounces.

On the 27th May, a little more than a fortnight after her discharge, she came to see me. There was no perceptible change in her symptoms.

The next time I saw the patient was on the 18th June, 1863, nearly thirteen months from the above date. I found her still somewhat stout, but less so than in the previous year. A month after leaving the hospital she had gone into service, but had done no heavy work. She remained in her place nine months, and then went home. She said the beating in the tumour was stronger than before, and she could sometimes "hear it whistle like a bird." She complained of great pain in the tumour at times, and of dyspnœa on exertion. She could lie down, and slept tolerably well. The tumour had extended upwards, and was very prominent just below the sternal end of the clavicle. At this spot its walls seemed very thin, and the pulsation was very strong. A double murmur was heard over the tumour, and more or less distinctly over the whole chest, both in front and behind. The second murmur was quite of a musical character; it was synchronous with the diastole of the heart, and was, I have little doubt, propagated from the aortic valves. The pulse was very feeble in the left arm, but good on the opposite side.

On the 19th July, she applied for readmission into the hospital, where she remained till the 18th August. During this time she complained much of dyspnœa and dysphagia, was unable to lie on her back, and had severe aching pain in the back and left side, and, at times, a hard ringing cough. She suffered much from vomiting, and took but little food. At her discharge the pulse was scarcely perceptible in the left wrist; the double murmur was audible, but the second portion was not musical as before; the breath-sounds at the back of the left lung were good.

She died at Runcorn, in Cheshire, on October 9th, 1863; and I am indebted to the late Mr. Wilson, of Runcorn, who sent the patient to me, and who watched her up to the time of her death, for a careful account of the autopsy, and for the parts involved in the tumour, which he removed. Speaking of her symptoms before death, Mr. Wilson says, "She did not die suddenly, but became gradually asphyxiated. For some days before her death the countenance was purple, the dyspnœa extreme, and the pulse imperceptible." The chief points of interest in the autopsy are these: the left pleura contained a large quantity of fluid, and the left lung was carnified; the pericardium also contained much fluid. There was a large aneurism connected with the arch of the aorta, commencing about two inches beyond the semilunar valves, and involving the whole of the arch to its termination, as well as about an inch of the vessel beyond. There were two distinct pouches in the aneurism; the first led between the third and fourth ribs of the left side, and presented, outwards, between them; the third rib was dissected and laid bare by the aneurism; the second pouch was between the first and second, and second and third left ribs; the second rib was completely dissected, and projected into this pouch, which presented opposite the second rib. The semilunar valves of the aorta were the seat of atheromatous deposit. The aorta just beyond the valves was dilated, and was the seat of atheromatous and calcareous degeneration. The mitral valve was healthy; the left ventricle was of normal thickness.

Remarks.—The length of time during which the disease existed after it had assumed a large size, without giving rise to any symptom of a very urgent, or distressing character, is an important and interesting feature in this case. There were no laryngeal symptoms; for a long time there were no violent attacks of dyspnœa; there was nothing, in fact, but the swelling, the pulsation, the pain, and the dyspnœa on exertion. I think this result must be attributed to the course the aneurism took—viz., forwards, pointing through the intercostal spaces. Taking this course, it exercised no great amount of pressure either on the trachea, or on the root of either lung, and thus there were no reflex spasms set up in those organs.

The peculiar musical character of the murmur was a phenomenon more interesting than important in a diagnostic point of view. I have occasionally heard murmurs of this kind in cardiac disease, but they are by no means frequent. The loudness of the murmur, so as to be at times audible to the patient, is a further point of interest. It is rare that patients hear their own cardiac, or aneurismal murmurs; and I think it is even still more rare that a murmur should be so loud as to be audible to a bystander, without the ear being brought either directly, or indirectly, through the medium of a stethoscope, in contact with the chest walls. One instance, and one only, of this kind I have met with. It was a case of disease of the aortic valves, and the murmur was so loud that it could be heard when the ear was placed within a foot of the chest; it is referred to in the chapter on chronic valvular disease.

CASE V.—*Aneurism of the Descending Thoracic Aorta becoming Diffused in the Abdomen. Death from Exhaustion. Autopsy.*—This case was a very remarkable one, and shows the curious course which a thoracic aneurism may take. It occurred in the practice of Dr. Lister, formerly of this town, and was seen by several medical men. I had frequent opportunities of examining the patient, and watched him up to the time of his death. I was also present at the autopsy.

In the spring of 1857 I was requested to see a man who had a tumour in the right iliac fossa. The history of the case was that, for some time previously (a few weeks), the patient had had severe pain, deep in the iliac fossa, and soon a tumour developed itself, and was found to pulsate. The tumour was thought by myself, and those who were present at the time I saw it, to contain fluid, and the supposition was that it was an abscess seated over the right iliac artery. At a subsequent visit it was resolved to pass an exploring needle into the tumour. This was accordingly done, when nothing but blood escaped. It now became evident that the tumour was either aneurismal or fungoid. It gradually increased in size until it occupied the whole iliac fossa, extending upwards as high as the crest of the ilium. It pulsated strongly. The patient became exceedingly feeble, and could scarcely be raised into the sitting posture for fear of syncope occurring.

From the general character of the tumour, as it grew larger, I expressed an opinion that it was aneurismal. At the same time, from the absence of bruit, which was never heard in front of the tumour, and from other circumstances, doubt was expressed, by some who saw the case, of the correctness of this diagnosis.

Within a day or two of the patient's death, on listening behind, a bruit was audible at one spot on the spine, about the commencement of the lumbar vertebræ.

The patient died on the 12th May, and the autopsy was made on the following day.

Autopsy.—On opening the abdomen, a large tumour was found projecting on the right side, pushing before it the peritoneum and right kidney; the ascending colon was pushed quite to the median line. The tumour filled the right iliac fossa, and extended upwards to the diaphragm, and backwards to the spine. It lay over the right external iliac artery, so as completely to conceal that vessel. The abdominal muscles, laterally and behind, were in close contact with the tumour, and formed its walls, so that they could not, at all points, be separated from that which formed the proper wall of the tumour. On cutting into the tumour, the external portion was found composed of laminæ of fibrine of somewhat recent formation. These laminæ, together with the peritoneum and condensed cellular tissue, constituted the sac of a very large diffused aneurism. The sac was filled with coagulated blood. The aneurism was connected with the thoracic aorta opposite the last dorsal vertebra; it communicated with the vessel by means of a square-shaped opening situated in the posterior part of the vessel. This opening led to a sac which had three distinct pouches, all lying above the diaphragm. On further examination it was found that the aneurism had passed beneath the ligamentum arcuatum internum, and behind the psoas magnus muscle, and had thus become diffused in the abdomen. The spot at which the original aneurism had burst could not be discovered. The sides of the bodies of the two upper lumbar vertebræ were partly absorbed. The posterior part of the abdominal portion of the aneurism was formed, in great part, by the quadratus lumbarum muscle. The tumour had caused absorption of the psoas magnus, and had dissected the lumbar plexus of nerves to a great extent.

There are certain diseases, especially, which may be mistaken for thoracic aneurism; these are, general dilatation of the aorta, disease of the aortic valves, and thoracic tumours. I have notes of some cases of dilatation of the aorta, in which the general symptoms much resembled those of aneurism; but it would occupy too much space to relate them. With reference, however, to cardiac disease and thoracic tumours, I will relate two cases of a somewhat instructive character. There can be no doubt that there is a great deal of difficulty, at times, in deciding whether a bruit, heard over the aorta, is connected with incipient aneurism, or disease of the aortic valves; but, as a rule, I think a careful consideration of all the features of the case will enable us, in a large majority of instances, to give a decided opinion.

Case VI.—I was consulted some years ago in the following case:—A gentleman, who had previously enjoyed good health, was seized with a good deal of pain about the sternum, throbbing in the neck, and a sensation of choking in the throat. He consulted two physicians, who expressed an opinion that the disease was aneurism of the aorta. They gave an unfavourable prognosis. The effect on the patient may be imagined: all his symptoms seemed to increase, he became uneasy and anxious, his spirits drooped, and his friends were much alarmed about him. He continued in this state for six or seven months, at which time I first saw him. I found no dyspnœa, no dulness behind the sternum or elsewhere, no evidence of pressure in any direction; but I found distinct evidence of aortic regurgitation—viz., a diastolic murmur heard at the base of the heart, and towards the right clavicle. There was the jerking pulse, so indicative of aortic regurgitation, with symptoms of slight hypertrophy of the heart. Rheumatic pains existed in different parts of the body, with symptoms of dyspepsia. I was able to assure the patient and his friends that I believed no aneurism existed, and that, formidable, undoubtedly, as the affection—valvular disease of the heart—was, yet, with due care, and proper attention to the general health, life might be, not only indefinitely prolonged, but made free even from much discomfort. It is several years since this opinion was given, and the results have not falsified the prognosis. The patient rapidly recovered his spirits; his rheumatic and dyspeptic symptoms were relieved by treatment; and having had explained to him the exact nature of his ailment, and the precautions that were necessary, he has been able to live with comfort, and with but little sensation of his cardiac malady.

The following case will serve to illustrate the symptoms which occasionally arise in connection with thoracic tumours, and which may lead to the supposition that an aneurism exists:

CASE VII.—Gustavus G., 63 years of age, was admitted into the Liverpool Northern Hospital, under my care, on the 14th March, 1863. He was a man of sallow complexion, and very unhealthy aspect. He said he had had difficulty of breathing, and of swallowing, for seven or eight weeks. He had suffered formerly from rheumatic fever. On making a physical examination of the front of the chest, I ascertained that there was no dulness on percussion, either over the sternum or at the upper part of the lungs. There was a distinct double murmur audible at the base of the heart and towards the right clavicle. The breathing was interrupted (wavy) at the right apex; on the left side it was good. The back of the chest was not examined. The patient was able to walk about, but had dyspnœa on exertion, and when he lay down. He was consequently propped up in bed. There was a good deal of fulness about the neck, evidently depending on obstructed venous circulation. On the 21st March, a week after admission, œdema of the right arm came on. A careful examination was made of the pulse in the two arms; it was thought that the left pulse was slightly fuller than the right. On both sides the pulse was jerking.

He continued with but little change up to the 26th March, on the morning of which day, as he was leaning out of bed, he died suddenly.

Autopsy.—The heart was much enlarged, and fatty on its surface. The left ventricle and the aorta were full of dark fluid blood; the pulmonary valves were healthy; the aorta was dilated and atheromatous; the aortic valves were the seat of firm, warty deposits; the mitral valve was healthy. *At the root of the right lung there was a mass of scirrhous glands,* which must have compressed the superior vena cava, and probably the innominate artery. The œsophagus, at its termination, was much thickened, and had a scirrhous appearance. The thyroid body was scirrhous; the gall-bladder contained forty-six gall-stones and no bile; the kidneys were healthy.

Remarks.—I had no difficulty, in this case, in deciding as to the presence of valvular disease of the heart. The symptoms pointed to aortic regurgitation; but the existence of dysphagia, the full condition of the vessels of the neck, and, especially, the œdema of the right arm, and the diminished volume of the right pulse, made me suspect that there was some thoracic tumour—an opinion which I expressed during the patient's life, without attempting to decide what was the exact nature of the tumour.

Case VIII.—*Aneurism of the aorta. Treatment by absolute rest. Great improvement. Discharge. Subsequent rupture of aneurism. Death. Autopsy.*—The treatment of internal aneurism by absolute rest has been strongly advocated by Mr. Tufnell, of Dublin; and he has recorded some very interesting cases, in which he adopted the practice successfully. In the following case, I carried out the line of practice he has recommended, which consists, essentially, in keeping the patient in the recumbent posture, and in administering a restricted diet. This treatment, as will be seen, was commenced after various other modes of treatment—moderate diet, iodide of potassium, ice to the tumour, acetate of lead—had been tried without any success.

John L——, a married man, 45 years of age, was admitted into the Liverpool Northern Hospital, under my care, on December 14th, 1864. He applied to the hospital in consequence of a pain in his back; but, on examination, a pulsating tumour was found below the right clavicle. He gave the following history.

For the previous seventeen years he had been a ship-keeper, on night duty, but, before that time, he worked hard as a labourer. He had been accustomed to drink freely. When he was about 28 years of age, his right arm was amputated for disease of the elbow-joint, following an accident. About three years before he applied to the hospital, he had a fall, by which he broke some of the ribs of the right side. For several years he had suffered from rheumatic pains in the limbs and stump. For twelve months before admission, he had occasionally been seized with violent attacks of sickness and cough; but he had never expectorated any blood. About seven or eight months before admission, he noticed a pain of a shooting character just below the right clavicle; and, about three or four months afterwards, he felt a pulsating lump in the same spot. This lump, he said, had grown very slowly, and was scarcely larger than when he first felt it. He seemed to attach but little importance to it, and considered the pain in the back his chief ailment.

Physical Condition, etc.—Below the right clavicle, opposite the lower margin of the second rib, a little external to its junction with its cartilage, there was a tumour, conical in form, with a rounded apex. The tumour had pushed the rib outwards, and appeared immediately beneath the skin. The visible portion was about an inch and a half in diameter. It had a soft fluctuating feel, as if containing fluid. It was the seat of pulsation, which was visible, expansile, heaving, and of moderate force. Pressure diminished the size of the tumour. Percussion revealed an area of dulness, bounded above by the clavicle, below by the third rib, internally, by the median line of the sternum, and, externally, reaching nearly to the nipple-line. There was normal resonance on the left side of the chest. Over the tumour, and below the middle of the right clavicle, a loud murmur was heard, synchronous with the systole of the heart. A soft systolic murmur was heard at the apex of the heart. The pulse was small, regular, and of equal volume on both sides. The patient was somewhat emaciated, had sharp features, and an anxious expression of countenance. He complained of severe pain along the back; and there was some tenderness at its lower part. There was no pain over the tumour. He had a severe barking cough, and dyspnœa on exertion; but there was no dysphagia. The veins of the left arm were rather large and knotted, but there was no œdema of the arm, nor of the stump. The digestive organs were unaffected. Both pupils were of the same size.

The patient, on admission, was put on a moderately spare diet, a small quantity of meat being allowed daily. The first remedy that was tried was iodide of potassium, which was given in large, and gradually increasing doses, until the quantity given was twenty grains three times a day. This treatment was continued for some time. No perceptible effect was produced on the aneurism; but the patient's health began to suffer, and the remedy was accordingly stopped.

After an interval of some weeks, the application of ice was tried. The patient was kept in bed, and a bag of ice was applied over the tumour during the day. This was continued for many days; but no noticeable change resulted in the aneurism.

On March 21st, three grains of acetate of lead, with half a grain of opium, were ordered to be taken, three times a day. This treatment was continued for a week, when symptoms came on which induced me to discontinue it.

On April 6th, in consequence of a good deal of pain in the chest being complained of, six ounces of blood were taken away by venesection. This was followed by a diminution of the pain.

On April 18th, the patient was ordered to confine himself entirely to his bed, and to keep in the horizontal posture. He had, up to this time, been in the hospital more than four months, and no perceptible change for the better, or worse, had, as far as could be observed, occurred. He was put on a very restricted diet; no stimulants were given, and, throughout the treatment, no medicines, except an occasional purgative or anodyne. These, however, were but rarely required. The man was intelligent, and tolerably manageable, and with one exception, when he walked from one ward to another during the time that the hospital was being cleaned, I believe he scarcely moved from the horizontal posture for a period of nearly eleven weeks. During this period, his health continued good; he slept well; complained of but little pain; had no sickness; and his cough became less frequent. His pulse (which, before the treatment was commenced, used to average from 80 to 90) fell to from 60 to 70. On one occasion, a few days before he was kept in bed, it was found as follows: Standing, 92; sitting, 84; lying, 70 per minute.

The diet which was ordered for the patient was, seven ounces of bread, three ounces of meat, and eight ounces of fluid, daily.*He was allowed small quantities of ice

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* Breakfast.—2 oz. bread and butter, 2 oz. cocoa in milk. Dinner, —3 oz. meat (boiled or roast), 3 oz. potatoes or bread, 4 oz. water or light claret. Supper.—2 oz. bread and butter, 2 oz. milk or tea.

to relieve his thirst, and one pipe daily. For some weeks after the treatment was commenced, more fluid than was ordered was taken, probably about a pint daily, but, subsequently, the above quantity (eight ounces) was rigidly adhered to.

Towards the end of May, it was very evident, from the diminished elasticity and pulsation in the tumour, that consolidation of the sac was taking place; and towards the middle of June there could be no doubt that this result had been produced. The patient was allowed to get up during the twelfth week of treatment. He was, however, kept in the hospital till August 12th, when he was discharged.

The following are the notes taken of the condition of the aneurism, etc., at the termination of the treatment.

"The tumour has diminished in prominence, in size, and in area of dulness. Pulsation is felt over the whole tumour; it is distant, and gives the sensation as if a good deal of solid substance existed between the skin and the inside of the tumour. A systolic murmur is faintly heard over the tumour, and at the apex a double knock is felt. In the apex or presenting portion, a very remarkable change has taken place: instead of giving the sensation, as at first, of a soft fluctuating swelling, very like that of an abcess, it is now hard, and feels like a solid mass. The pulsation, at this spot, where it was originally most marked, is now least so, and can only be discovered on careful examination."

After the patient left the hospital he came to show himself, from time to time. He resumed his work as a ship-watcher, and said he felt quite equal to it. He scarcely complained of the pulsation in the aneurism. I have the following notes of his condition on the 11th of November, viz., three months after his discharge:

"He says he is well, and that he feels the beating very little. It is only occasionally that he perceives it. The tumour is less prominent than when he went out; it is smaller, and harder. There is no pulsation at the apex."

So far the case had progressed satisfactorily, and I think it highly probable that, if the man had been in that position of life which would have enabled him to take sufficient care of himself, he might have lived, even, for several years. He died, suddenly, at his home on the 5th of March, seven months after leaving the hospital, and a *post-mortem* examination of the body was made the following day.

An aneurism was found connected with the arch of the aorta. It projected four inches to the right of the sternum, and extended from the third to the sixth rib. A large quantity of blood was found in the right pleura, into which the aneurism had burst.

The anterior part of the aneurism was filled with hard firm laminæ of fibrine, more than an inch in thickness; but the posterior wall was thin, and the rupture had taken place there. There was slight calcareous deposit on the mitral valves; otherwise the valves of the heart were healthy. The substance of the heart was somewhat fatty.

I now pass on to make a few remarks with reference to the diagnosis and treatment of aneurism.

Diagnosis.—In all cases of aneurism of the arch of the aorta—the part which is most frequently attacked with the disease—I believe that the first least equivocal sign we should look for, is, a dulness opposite the upper part of the sternum. It is there that the tumour first begins to push aside the lungs, and to come in contact with the thoracic walls, and it is, usually, from this spot that the dulness extends either to the right, or left side, according to the direction which the aneurism takes. This dulness may, it is true, be caused by a non-aneurismal tumour; and further, it may, when slight, be the result of a general dilatation of the aorta; for, in one case which I met with, some of the symptoms of aneurism were present, and amongst them was a slight dulness opposite the upper portion of the sternum, but not extending on either side. In this case there were well-marked symptoms of aortic regurgitation, and I had no difficulty in concluding that there was valvular disease of the heart, with hypertrophy. There was some doubt, however, in my mind whether the dulness was due to an aneurism of small dimensions, or a dilated aorta. The general symptoms made me incline to the latter view. Through the kindness of the gentleman under whose care the patient died, I ascertained the result of the *post-mortem* examination, and had an opportunity of seeing the heart, and a portion of the aorta of the patient. There was no aneurism. The heart was hypertrophied, and the aortic valves were incompetent. The aorta was very much dilated and atheromatous.

Bruit.—With regard to the existence of a bruit in aortic aneurism, some diversity of opinion has been expressed. Guided by the result of their own experience, physicians have expressed their views, according as a bruit has predominated in their cases, or the contrary. Looking back at the cases of aortic aneurism which I have seen, both in my own practice, and in that of others, and of which I have made notes, I find that, in the majority, no bruit was audible. In two of the fatal cases I have recorded, there was no bruit; so that although the existence of a bruit may assist us in our diagnosis, its absence is no proof whatever of the non-aneurismal nature of a pulsating tumour.

State of the Pupils.—The contraction of the pupil, as a consequence of thoracic aneurism, is a symptom which has only of recent years attracted attention. The fact, which this symptom indicates, is, pressure, or irritation of some kind, of the sympathetic nerve, and would occur in any case of thoracic tumour, and not simply in aneurism. Still the knowledge of the symptom, and of its cause, is by no means unimportant, for without this knowledge we might be misled as to the nature of the symptom, and be unable to explain its occurrence. I have observed it, as a permanent symptom, more especially in aneurisms at the root of the neck; in some cases, I have seen the pupil on the aneurismal side only occasionally contracted, whilst, in others, there has been no difference in the size of the two pupils.

Treatment.—The treatment which I have adopted has been chiefly directed to two ends: 1st. To promote the formation of fibrinous deposits in the aneurismal sac; and, 2ndly, to improve the nutrition of the arterial coats.

In the production of the first object, the formation of fibrinous deposits in the aneurismal sac, quiet is, no doubt, one of the most important points to be insisted on; and it is remarkable how much better patients often become, when they are admitted into hospital, from this simple cause; and in considering the effect of drugs in favouring coagulation in an aneurism, we must not forget to allow its due value to the rest which we prescribe. In some cases, I have extracted blood in small quantities, from time to time, but not to the extent of weakening the patient. With regard to diet, I have always allowed a small quantity of meat daily, under the impression that, by keeping the blood at a fair standard, I was taking the best means of producing a deposit of fibrine in the aneurismal sac, as well as of improving the general nutrition.

But I must say a few words with reference to the treatment by prolonged rest, such as I adopted in case No. 7. The results which followed the treatment must, I think, be considered satisfactory, and, the more so, that various other measures—moderate rest, iodide of potassium, ice to the tumour, acetate of lead—had been tried without success. It is difficult to get this kind of treatment properly carried out, especially in hospital practice. Great moral courage is required on the part of the patient, for it becomes very irksome to lie for many weeks in the horizontal posture, as is necessary, in order to give the plan a fair trial. The patient should never rise from this posture, and the bed on which he lies must be prepared with this view. The effect produced on the circulation by the recumbent position is well known, and must have a material influence in promoting the formation of fibrinous deposits in an aneurismal sac. In my patient the pulse fell 10 to 20 beats per minute. In the course of five or six weeks from the commencement of the treatment, it was evident that the interior of the sac was becoming consolidated. The *post-mortem* examination showed that fibrine had been deposited in large quantities, and had become very firm; but, unfortunately, this deposit was not complete all over the sac, and the habits of life of the patient were not favourable to that quietude of the circulation, which is so especially desirable in all aneurismal diseases. Looking, however, at the results of the case which I have detailed, and of those which have been brought forward by others, I think the plan of prolonged rest, and somewhat scanty diet, is deserving of more extended trial.*

With regard to the use of medicinal substances, given with the view of producing fibrinous deposits, I must say that I have but little confidence in their value. Iodide of potassium, in large doses, has been strongly recommended, but my own experience of its use does not enable me to say much in its favour. I have given it on several occasions, and have been disappointed in its effects; but in the hands of other practitioners, it seems, in son instances, to have been useful. Again, I have tried aceta of lead, but this, also, has failed, in the cases in which have used it, to produce any apparently beneficial effect.

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* I am at the present time (Feb., 1868) trying this plan in a case of aneurism of the abdominal aorta; but the patient has only been under treatment for a few days.

In attempting to carry out the second object of treatmen viz., to improve the nutrition of the arterial coats, we mu bear in mind that atheroma is the predisposing cause most, if not of all aneurisms connected with the gre blood-vessels. All such measures, therefore, as may te to check this degenerative change should be resorted t and with this view I have given iron and other tonics, i some cases, apparently, with great benefit.

CHAPTER VII.

ON THE USE OF ALCOHOLIC STIMULANTS.

WHATEVER may be our views of the physiological action of alcohol, our belief in its therapeutic value rests on no mere speculative notions, but is supported by the testimony and the experience of almost all practitioners of medicine. As is the case with many other remedies, however, our knowledge of its action is imperfect, and we stand much in need of correct principles for its administration. Its general applicability to various conditions of the system, as a health-restorer, has been almost universally recognized; and in all ages, physicians have resorted to its use. It is only, however, of late years, that it has risen to the position of one of the most important, and most freely used, of our therapeutic substances; and that its employment has been sanctioned, and its value demonstrated, in numerous diseases, in which, it was previously considered not only useless, but highly injurious. Both in medical and surgical practice, there can be no doubt that, for some years past, a larger quantity of alcoholic liquids has been used, than during any other equal period in the history of medicine in this country. The statistics of our hospitals would at once prove this. As to the influence it has had on the rate of mortality in these institutions, I cannot now stop to inquire. It would be very satisfactory, if statistics on a large scale could be drawn up, which would show the rate of mortality, and the duration of diseases, during two given periods—one, before, the other, since the present system of treatment has become more general.

But, whilst unable to test the effects produced by alcohol by a direct appeal to statistics, I may be allowed to offer an opinion on its use, founded on a careful observation, for some years past, of cases, both in hospital and private practice. Speaking generally, I believe it may be said, that the early administration of alcoholic stimulants has reduced the rate of mortality of some diseases; and that, in a far larger number, it has diminished the intensity of the attacks, and accelerated the convalescence of the patients.

I have not arrived at these conclusions without careful study, and observation; I have seen alcohol used, and I have used it myself, largely. I have given it under circumstances when I could carefully watch its effects; and I have noted the influence it has produced both in acute and chronic diseases.

And here I would lay down a general principle, which is, I think, applicable to all remedial agents, viz., that the therapeutic value of alcohol does not rest on its supposed use as an article of diet, nor on any supposed chemical change it may undergo. It would be very satisfactory to know, by incontrovertible evidence, that it is used up in the system, either as a heat- or fat-producer, or in affording nourishment to the nervous tissue. The establishment of such a fact, on a sound basis, would increase our estimate of the usefulness of alcohol; but its value as a therapeutic agent rests on no such assumption, and may be altogether independent of any such action. We may grant all that some chemists have, of late, advanced; that the substance is not oxidized in the system; that every particle of it is eliminated unchanged; that, contrary to our long cherished notion, it does not form fuel for the combustion going on in the body; but we do not, by all this, in anywise impugn its therapeutic value, nor is our confidence shaken in its applicability to various conditions of the body, even when taken as an article of habitual use.

Amongst the most important, if not the most important, of the actions of alcohol, in a therapeutic point of view, is the influence which it produces on the circulating system. That the administration of alcohol increases the power of the heart, diminishes the frequency of its beat, and augments the force of its contraction, under conditions of debility, are facts which must be familiar to all who are in the habit of prescribing it. And there is nothing more striking, and, I will add, more satisfactory, than to watch the effects of the remedy in those cases of inflammation, which are denominated asthenic, and which are marked by a rapid and weak pulse. Under its use we see a pulse, abnormally quick, gradually fall; we see an irregular one become steady and regular; we see delirium subside, and a tongue, foul, dry, or brown, assume a moist, and healthy character.

But the effects which alcohol produces on the circulation cannot be accounted for, I think, simply on the ground that it acts as a stimulant and tonic to the heart. It has been proved by some physiologists, that the introduction of certain substances into the blood has a tendency to increase the rapidity of the circulation, whilst the introduction of others has a directly opposite effect. Amongst the latter is alcohol; and, according to the observations that have been made, when this substance is injected into the vessels of a living animal, there is a retardation of the circulating current, apparently from some physical influence which the alcohol produces, either on the blood itself, or on the coats of the vessels, or on both. And

I would here remark, that when alcohol is taken into the stomach, although we have evidence of increased action of the heart, this by no means proves that the blood is circulating more rapidly through the capillaries of the body. The very symptoms, indeed, which follow the administration of a small dose of alcohol—namely, a sensation of fulness and heat of skin—and the congestion which ensues when the dose is increased, tend to prove that there is some impediment to the peripheral circulation. Such a state of capillaries must, necessarily, give rise to an increased tension of the arterial system, and a fulness of the pulse; especially when taken in connection with the effects produced on the heart itself by the action of alcohol.

It appears to me, that a knowledge of this local action of alcohol on the capillaries, serves to explain, in some measure, the effects which we witness, in certain cases of disease, from its administration.

Is it not true, that, in certain wasting diseases, in various forms of acute inflammation, where there are rapid changes going on in some particular organ, or part—a rapid development of inflammatory products, of serum, of lymph, of pus—under the influence of alcoholic liquids, these changes are checked, and the wasting processes are arrested?

Now, there can be no doubt that, in these cases of inflammation, the vital changes of the part involved are *too active*. I do not say that the vital powers are *too high*—that there is *increased vitality;* but that the molecular changes of the part are *too rapid*.

When, in such cases, we administer alcohol, we often see a rapid arrest of these changes, and an absorption of the effused products; and I cannot but think that this result is due, not simply to the general influence of the remedy in improving the nutrition of the body, and in giving increased power to the heart; but, also, to its local action on the capillaries and tissues of the affected part.

It is very remarkable to see the large quantities of alcoholic liquids—wine, brandy, etc.—that are borne in disease, even by persons who have previously taken, whilst in health, little or none, without any of the ordinary physiological effects being produced—without the occurrence of intoxication, of headache, of furred tongue, or of disturbance of the stomach.

In the preceding chapters of this work, I have given many instances of the effects produced by alcohol in acute thoracic diseases, and of the rapid convalescence from these affections, which followed the liberal administration of wine, or brandy. It would take me far beyond my limits were I to consider the effects produced by alcohol in certain chronic affections; but I cannot dismiss the subject without one or two remarks as to its ordinary use.

From the influence which it exercises in certain states of disease, indications may be gathered for its use in conditions of health. From the various circumstances of constitution and habits of life, the moderate use of alcoholic beverages appears, in many persons, to be attended with great benefit. Those who argue against the proper use of alcohol, from the ill effects produced by its abuse, appear to me to miss the question entirely; and they might, on like grounds, attack the use of tea, or coffee. No doubt, the highest development of physical strength can be obtained without the use of spirits in any form whatever. Could we, indeed, all live the life of the athlete—breathe pure air, take exercise largely, and keep our digestive organs in a high state of efficiency—we might dispense with alcoholic liquids. Whilst, however, men lead sedentary and laborious lives, working their nervous system, and breathing an atmosphere, itself depressing; whilst the organs of digestion are scarcely able to perform the work which is required of them, in order to supply the waste of tissue which is going on; whilst, even constitutionally, the circulation is languid, and the assimilative processes are weak — a substance, like alcohol, dietetic or medicinal, whatever we may call it, may be used in moderation by many, not only without disadvantage, but with decided benefit. And in forming an opinion of the value of alcohol, we must never lose sight of the fact, that just as the pathological conditions of certain acute diseases modify its physiological action, so, the various constitutional defects which, in a large proportion of the population, detract from the standard of perfect health, may be indications for its habitual temperate use.

But, in expressing my opinion of the value of alcohol as a therapeutic agent, and as an article of diet, I would warn against the danger we may run of using it indiscriminately, and I would protest against any extremes of practice, or careless administration of the remedy, without due regard to the pathological conditions to which it is applicable. I consider alcohol, properly used, one of the most powerful of therapeutic agents. It is a remedy which has no special application to any one disease, but it may be used beneficially in a large number of diseases, under certain circumstances. In administering it, we must be guided, not by the name of the affection we have to treat, but by the symptoms which are present. Alcohol has no restricted local action; its effects are not confined either to the nervous, or vascular system; but it has, undoubtedly, great power in rousing the flagging action of the heart, and in restoring, in some cases, the perverted function of the brain.

There can be no doubt, I think, in the minds of those who have watched the effects of alcoholic stimulants, of their therapeutic value in some of the acute diseases of the chest. In the bronchitis of the aged, attended as it often is with profuse secretion, threatened apnœa, and a weak pulse, wine or brandy, given at regular intervals, is, in my opinion, the most useful remedy we possess. Again, in pneumonia occurring in aged people, stimulants may be given without any hesitation. I attended, in the winter of 1866, with Mr. Blower, of Everton, a lady in her 80th year, who had acute inflammation of the left lung. The attack was very severe, and three-fourths of the lung became involved. So urgent were the symptoms on the second day I saw her, that we held out but little hope of an improvement being effected. However, we increased the quantity of stimulants which the patient was taking, and we ordered a table spoonful of brandy to be given every hour. Under this treatment, which was combined with as much nourishment as the patient could take, she began to improve. The twelve ounces of brandy, daily, were continued for several days, without producing any other effect than that of bringing down the pulse, and improving all the pneumonic symptoms. There was no disturbance of the sensorium, no intoxication, and no interference with the functions of the stomach. At the end of a week the patient was out of danger, and I ceased my attendance. She perfectly recovered.

I might refer to other cases of a like nature, showing the value of alcoholic stimulants in the treatment of the aged; and it is not simply at the one extreme of life that such treatment is applicable. In the treatment of the pulmonary affections of children, I have found stimulants of great value. According to my experience, children bear stimulants very well. I have seen the treatment of infantile pneumonia, and of bronchitis, by tartar emetic and mercury, but I have been led to form an unfavoural opinion of the value of these drugs in such cases, and, my own practice, I have adopted a moderately stimulati treatment, which I have found followed by satisfacto results.

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